BRAIN.

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Original Articles and Clinical Cases.

HYPNOTIC AND POST-HYPNOTIC APPRECIATION OF TIME; SECONDARY AND MULTIPLEX PERSONALITIES.

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I COMMENCED to employ hypnotism as a therapeutic agent in 1889, and in less than two years treated over 500 patients; of these 48 per cent. became somnambules, *i.e.*, were unable when hypnosis terminated to recall the events of hypnotic life. Having observed that the curative effect of suggestion was increased by prolonged hypnosis, I frequently suggested to my patients that they were to remain in that state until a given hour—usually that of their next meal—and then left them. Although rarely present at the conclusion of the experiment, I obtained ample and trustworthy evidence that hypnosis invariably terminated at, or within a few minutes of, the hour indicated, and thus accidentally discovered that deep hypnosis was associated with an increased appreciation of time. These observations—the by-product of therapeutic work—led to more careful experiment with somnambules, the majority of whom were males and all in good health. The following were the usual suggestions given: (1) A simple act was suggested during hypnosis which was to be carried out at a given time

before that state terminated. (2) The subject was told during hypnosis that this state was to terminate at a specified future hour. (3) The performance of a simple act at a given hour after the termination of hypnosis was suggested. (4) Awakening from natural sleep at a given hour was suggested during hypnosis. (5) The subject was told in the waking state that he was to pass into the hypnotic condition at a given hour, remain hypnotised for a specified length of time and perform certain simple acts at stated intervals; then pass again into the normal state and remain in it for a specified time, and again pass into the hypnotic condition. These experiments, continued from 1889 to the present date, have been frequently repeated before competent observers. The majority of the suggestions were executed at the moment indicated, while in the remainder the error in time appreciation rarely exceeded five minutes. In every instance, except those referred to in group No. 4, the subjects were carefully watched from the beginning to the end of the experiment.

Similar phenomena have been observed by nearly all who have done practical hypnotic work, and I have seen experiments resembling those just cited reproduced in various foreign cliniques. Two, formerly regarded as the most remarkable of their kind, were made by Beaunis and Liégeois. In one of these a visual hallucination appeared after a suggested interval of 172 days, and in the other after 365. The late Professor Delbœuf, however, pointed out in reference to all such experiments that even in the longest a fixed date had been impressed upon the subject's mind. Thus, Beaunis' subject was told that the 172nd day was New Year's Day, and Liégeois' was impressed by the fact that the suggestion was to be executed in a year from the time it was given. The experiments, therefore, did not involve the carrying out of a suggestion after the lapse of so many days, which the subjects were supposed to count as they passed, but simply on the arrival of a fixed and easily recognised date. This objection applies with great force to my earlier cases. In every instance a specified hour was suggested; and, in order that the subjects

might be conveniently watched, the time involved rarely exceeded a few hours. This point will be again referred to in treating of the theoretical explanation of the appreciation of time. Meanwhile, I wish to draw attention to the experiments made by Delbœuf with the object of eliminating an easily recognised fixed date from the suggestions.

Delbœuf's Experiments.

These occupied a week, from Saturday, October 2, to Saturday, October 9, 1886. His subjects were his two maid-servants, J. and M., sisters, aged respectively 20 and 23. All the experiments were of a similar character; from time to time during hypnosis the subjects were told that they were to do something at the expiration of a certain number of minutes, an interval of waking life always intervening between the suggestion and its fulfilment:—

*Exp.** 1.—Sat., Oct. 2, 1886; subject J.; time 6 a.m. *Sug.:* At the expiration of 350 m. J. was to ask Delbœuf if she should harness the donkey. Delbœuf's wife was ill and went out in an invalid carriage drawn by a donkey, but J. had nothing to do with it; thus her question would be unusual. *Res.:* The impulse to ask the question came into J.'s mind at the time it was due, but she successfully resisted it.

Exp. 2.—Subject M.; time 8 a.m. *Sug.:* At the expiration of 350 m. M. was to ask Madame Delbœuf if she would like to go out. Under the circumstances the question would be an unusual one. *Res.:* At 1.50, the hour indicated, M. was impelled to ask the question, but as she happened to be in the village on an errand, was unable to do so. She returned at 2.30 and carried out the suggestion.

Exp. 3.—Mon.; subject J.; time 9.15 a.m. *Sug.:* In 900 m., *i.e.*, at 12.15 a.m., J. was to go into the bedroom of one of Delbœuf's children and pull his ear. *Res.:* Suggestion carried out 95 m. too soon.

Exp. 4.—Subject M.; time 9.25 a.m. *Sug.:* M. was to embrace Mlle. H. Delbœuf at the expiration of 700 m. *Res.:* Twenty-five m. before the suggestion fell due M. looked for Mlle. H. Delbœuf in order to embrace her, but could not find her.

* Exp. = Experiment. Sug. = Suggestion. Res. = Result. d. = days. h. = hours. m. = minutes.

Exp. 5.—Subject M.; time 10.4 p.m. *Sug.:* In 900 m. M. was to embrace Mlle. C. Delbœuf. *Res.:* At the time indicated, *i.e.*, 1.4 on Tuesday afternoon, M. was having her lunch with the other servants when she suddenly got up from the table, sought and found Mlle. C. Delbœuf and carried out the suggestion.

Exp. 6.—Tues.; subject J.; time 6.30 a.m. *Sug.:* At the expiration of 1,600 m. J. was to pull the cook's nose. *Res.:* Suggestion carried out 60 m. too soon.

Exp. 7.—Subject M.; time 6.45 a.m. *Sug.:* At the expiration of 1,150 m. (Wed., 1.55 a.m.) she was to go into the cook's bedroom and pull her by the ear. (The cook was told what was likely to happen.) *Res.:* At the exact hour the subject had a strong impulse to carry out the experiment, but resisted this until 4.15 a.m., when she gave in and went to the cook's room. The cook laughed, whereupon M. said: "If you laugh I shall pull your ear."

Exp. 8.—Wed.; subject J.; time 9.55 a.m. *Sug.:* At the expiration of 1,300 m. (Th., 7.35 a.m.) J. was to ask Madame Delbœuf if she would like to have her hair dressed. *Res.:* Suggestion carried out 69 m. too soon.

Exp. 9.—Subject M.; time 6.55 a.m. *Sug.:* At the expiration of 1,500 m. (Th., 7.55 a.m.) M. was to ask Madame Delbœuf if she required anything. *Res.:* Suggestion carried out with absolute accuracy.

Exp. 10.—Friday; subject M.; time 6.30 a.m. *Sug.:* M. was to feel sleepy at 10 p.m.; go to bed and sleep profoundly. *Res.:* Correct.

Exp. 11.—Subject J.; time 9.15 a.m. *Sug.:* At 11 p.m. J. was to go into M.'s room and give her a complicated suggestion to be carried out at 5.30 next morning. *Res.:* J. went to bed at 10 p.m. in her own room and quickly fell asleep. At 10.50 p.m. (10 minutes too early) she went to her sister as suggested, but could not afterwards recall what she had said or done.

Exp. 12.—Subject M. *Sug.:* M. was to execute the orders just referred to at 5.30 a.m. She did nothing, however, beyond carrying out the first part of the suggestion, *i.e.*, that she should go to bed and sleep profoundly, and was unable to remember whether her sister had said anything to her or not.

Exp. 13.—Sat.; subject J.; time 9.30 a.m. *Sug.:* At the expiration of 3,300 m. J. was to ask Delbœuf if she should carry a small ladder to the pear tree. *Res.:* The impulse to ask the question arose 90 m. too late, but J. did not give way to it.

Exp. 14.—Subject M.; time 10 a.m. *Sug.:* Similar to the last to be executed after a like interval. *Res.:* Identical with the above, but 90 minutes too soon.

Summary.

There were in all fourteen experiments. The suggestions, to be carried out after the lapse of 350, 900, 1,600, 1,150, 1,300 and 3,300 minutes respectively, were made at varying hours of the day and night, while some fell due at night after the lapse of several days.

Results.

Three of the suggestions were fulfilled at the moment they fell due; four were carried out, but not at the exact time. In three an impulse to carry out the suggestion arose at the right moment. In one of these, the subject successfully resisted the suggestion, in another, she was accidentally prevented from executing it, while in the third, she struggled against it for two hours and twenty minutes and then carried it out. In three, the impulse to carry out the suggestion arose, but not at the correct time. In one of these cases accidental circumstances alone prevented the suggestion from being carried out, but in the two others, the subject successfully resisted it. One alone, No. 12, failed completely, but this may have been due to the fact that No. 11 was not fulfilled in its entirety, *i.e.*, it is doubtful whether J. gave the requisite orders to M. As, however, the essential fact in the experiments was not the actual carrying out of trivial and sometimes absurd suggestions, but the patient's recognition of the terminal time, the cases in which an impulse arose at the correct moment must be classed amongst the successes. The number of these is thus raised to six, while of the eight remaining experiments seven were partially successful. Of the latter four were carried out and an impulse to execute the suggestion arose in the other three, but in none was the time accurate; the error varied from a tenth to a thirty-seventh of the interval.

Remarks.

J. and M. were strong healthy peasant girls, who had frequently been hypnotised and were both good somnambules. At an earlier date, Delbœuf had made the following experiment with J. After explaining what he proposed to do, and obtaining

her consent in the waking state, he hypnotised her, extended her arms on a table, and suggested that the right should be insensible to pain. Each arm was then burnt with a red-hot bar of iron, 8 millimetres in diameter, the extent and duration of its application being identical in both, but there was pain in the left arm alone. The burns were bandaged and J. sent to bed. During the night the pain in the left arm continued, and next morning there was a wound on it, 3 centimetres in diameter, with an outer circle of inflamed blisters. On the right there was only a defined eschar, the exact size of the iron and without inflammation or redness. The day following the left arm was still more painful and inflamed; analgesia was then successfully suggested, when the wound soon dried and the inflammation disappeared.

Later J. married and was 29 years of age when her first child was born. She was attended by Dr. Fraipont, Lecturer of Gynæcology at the University of Liège, while Delbœuf induced hypnosis and made suggestions of analgesia. When J. was aroused from the hypnotic state, after the birth of the child, she was unable to recall anything that had occurred.

J. and M. were very imperfectly educated and could with difficulty tell the time by the clock. It was impossible for them to at once reduce such a large number of minutes as 400 into hours and they were obliged to proceed by successive additions, thus:—1 hour = 60 minutes; 1 hour and 1 hour makes 60 + 60 = 120; 120 + another hour makes 180, and so on. Before reaching 360 they had often made mistakes, and, no matter what method they adopted, were absolutely incapable of reducing such numbers as 1,600 and 1,150 minutes into hours. Further, supposing they recalled the suggestion on the termination of hypnosis and noted what o'clock it was—which they neither did nor could do—they would still, for example, have to determine by mental calculation the hour which corresponded to 6.45 p.m., increased by 1,150 minutes, a feat entirely beyond their powers.

Later, in order to discover whether J.'s hypnotic arithmetical powers exceeded her normal ones, Delbœuf put the following problems to her during hypnosis:—(1) "Turn 350 minutes into hours." Answer: "Six hours. No, five and a half hours." (2) "Turn 1,200 minutes into hours." A. "Fifteen hours. No, twelve and a half hours." (3) "Turn 150 minutes into hours." A. "Two and a half hours." (4) "Turn 240 minutes into hours." A. "Two hours." "Think!" Answer, after two minutes, "Four hours." (5) "Turn 300 minutes into hours." Answer, after five seconds' calculation, "Four and a half hours." Then,

after a further calculation, "Five and a half hours." Finally, "Five and three quarter hours." Delbœuf asked J. to explain the last calculation to him. She replied: "This makes five and a half hours plus 3 times 10, which makes five and a half hours exactly." Delbœuf tried to help J. as follows:—"How much is 60 + 60?" Answer, "120 minutes." "120 + 60?" A. "180 minutes." "180 + 60?" Answer, after a long hesitation, "240 minutes." "240 + 60?" A. "290 minutes." J. had spent ten minutes over attempting to solve the last problem and showed great signs of fatigue. Delbœuf did not consider it necessary to push the questions further. It is to be noted that M. was a somewhat better arithmetician than J., and that, in the experiments referred to, her results were more accurate than J.'s.

Further Time Experiments.

The following were inspired by Delbœuf's, and the results were so remarkable that I feel justified in giving as many details as possible, in order to present a fairly complete picture of the subject of the experiments, and of the circumstances under which they were carried out:—

Miss A., aged 19, was sent to me by Dr. de Watteville for hypnotic treatment, on Sept. 2, 1895. Her health had been good up to twelve months previously, when violent muscular tremor of the right arm and hand commenced. After a few weeks this spread to the right leg, and then to the left arm and leg; the tremor ceased during sleep, but only then, while walking was always difficult and painful. There was almost constant diffused headache. During the last four months, there had been frequent attacks of pain in the region of the heart. After the patient had looked at a bright object—especially anything blue—everything else appeared of that colour; this impression persisted for about an hour, while its disappearance was always sudden and accompanied by a feeling of faintness. Menstruation began at 11, and was always painful, and there had been obstinate constipation from infancy. Her illness began after over-exertion and mental strain, the result of nursing a relative who died. There had been no previous hysterical symptoms and the family history was good.

Treatment.—Electricity, massage, careful drugging and change of air.

Result.—No improvement.

I succeeded in inducing fairly deep hypnosis at the first attempt; and this was repeated on September 4, 5, and 6, when the tremor almost entirely disappeared. From then to October 30 she was hypnotised on seven occasions. *Res.:* No tremor or other morbid symptoms. Bowels regular. Last period free from pain. Fourteen pounds gained in weight since beginning of treatment. No abnormal colour perceptions. Returned to work.

Remarks.—Hypnosis became more profound each time it was induced, and at the seventh séance Miss A. reached the stage of somnambulism, *i.e.*, she was unable in the normal state to recall the events of hypnotic life. From this date she could at once be made analgesic or anæsthetic by suggestion; touching the cornea or tickling the back of the throat with a feather produced no reflex, and the passing of a needle deeply into the flesh was unattended by pain.

During treatment suggestions had been made fixing the hour at which Miss A. was to fall asleep at night and the moment at which she was to wake in the morning. As these were remarkably successful, it occurred to me that she might prove a good subject for experiments similar to those of Delbœuf. Miss A. was an intelligent girl who had received an ordinary School-board education, and her arithmetical powers were in keeping with this; she could do ordinary sums in multiplication and subtraction with the aid of a pencil and paper, but failed, unless they were extremely simple, to solve them mentally. Notwithstanding this, she asserted that she had been the best in her class at mental arithmetic. She possessed no particular aptitude for appreciating the passage of time.

Exp. 1.—Nov. 5, 1895; time 4 p.m. Suggestion given during hypnosis: At the expiration of 5 h. and 20 m. Miss A. was to make a cross on a piece of paper, and write down the time she believed it to be without looking at clock or watch.—*Res.:* The suggestion was carried out the minute it fell due. *Remarks:* On this occasion I did not say anything to Miss A. about the experiment, either before or after hypnosis, and, being a somnambule, she retained in her waking consciousness no recollection of the suggestion. I told her mother its nature, but not the time at which it should be fulfilled. At 9.15 the same evening her mother noticed that Miss A. was restless, and asked her what was the matter. She replied, "I feel I must do something, but cannot tell what." At 9.20 p.m. she rapidly made a cross with a pencil and wrote "20 minutes past 9" on a piece of paper, at the same time saying, "It's all silliness." There was no clock in the

room, but her mother went into the next room where there was one, and found that the time was 9.20. When I again saw Miss A., I explained the nature of the experiments I proposed making, and instructed her to carry a pencil and paper during the day and to put them by her bedside at night. I did not describe the experiments as anything extraordinary, but simply told her that hypnotised subjects were often able to appreciate time, and that I wished to see whether she could do so. No pecuniary or other reward was promised or given. I told her I should make these suggestions from time to time, but not on each occasion she visited me. I neither told her in the waking condition that suggestions had been made, nor informed her relatives when I made them, nor what they were. They knew that suggestions of this nature were given frequently, but only became acquainted with them by seeing Miss A. carry them out, or by hearing from her that she had done so. Before making the suggestions, I wrote them down in my case book, and, when Miss A. again visited me, I copied into it what she had written on the different pieces of paper. In many instances I did not calculate when the suggestions fell due, and in others the calculations I made at the time were proved to be erroneous, the results of the experiments in these cases being only determined when the series was completed.

The experiments which followed were all of the same character, *i.e.*, during hypnosis Miss A. was told that, at the expiration of a certain number of minutes, she was to make a cross and write down the hour she believed it to be without consulting the clock, an interval of waking life always intervening between the suggestion and its fulfilment. The simple and uniform character of my experiments is due to the fact that Delbœuf's subjects resisted suggestions that were distasteful to them. The idea of making a cross on a piece of paper excited no opposition in Miss A.'s mind, while the fact that she recorded in writing the time at which the suggestion was fulfilled, especially when this was witnessed by others, put me in possession of evidence of a certain value. The arithmetical problems involved in the first one or two of the following experiments were comparatively simple. In No. 3, for example, as Miss A. could easily tell when 24 hours fell due, the suggestion practically resolved itself into one to be fulfilled in 100 minutes. Soon, however, the experiments became complicated and involved much more difficult problems in arithmetic.

Exp. 2.—Nov. 28, 1895; 2 p.m. *Sug.:* to be fulfilled in 320 m. *Res.:* Correct. *Remarks:* The suggestion was carried out

at 7.20 p.m. when the patient was in a friend's house. She had no watch with her and the clock in the room was wrong.

Exp. 3.—Dec.4; 3.15 p.m. *Sug.:* in 24 h. and 100 m. *Res.:* Correct. *Remarks:* When in a friend's house the following afternoon she carried out the suggestion at 4.55. She then asked the time. Her friend looked at her watch and told her, whereupon she remarked, "Your watch is 3 minutes fast." This was the case.

Exp. 4.—Dec. 12; 3.20 p.m. *Sug.:* in 24 h. 1,440 m. *Res.:* 3.20 p.m. Sat., Dec. 14: Correct.

Exp. 5.—Wed., Dec. 18; 3.45 p.m. *Sug.:* in 24 h. 2,880 m. *Res.:* 3.45 p.m., Sat., Dec. 21: Correct.

Exp. 6.—Tues., Dec. 24; 2.55 p.m. *Sug.:* in 30 h. 50 m. *Res.:* 9.45 p.m., Wed., Dec. 25: Correct.

Exp. 7.—Tues., Dec. 24; 3.10 p.m. *Sug.:* in 7,200 m. *Res.:* 3.10 p.m. Sun., Dec. 29; Correct. *Remarks:* When No. 7 was fulfilled the patient was teaching a Sunday school class, when she suddenly felt an impulse to make a cross and mark the time. It was only after doing so that she looked at the clock, which was behind her.

Exp. 8.—Tues., Dec. 31; 3.45 p.m. *Sug.:* in 4,335 m. *Res.:* 4 p.m. Fri., Jan. 3, 1896: Correct.

Exp. 9.—Dec, 31, 1895; 4 p.m. *Sug.:* in 11,525 m. *Res.:* 11.5 a.m., Wed., Jan. 8: Wrong. *Remarks:* The result ought to have been 4.5 p.m., Jan. 8. I re-hypnotised Miss A. on that day and asked her to recall the suggestion I had made on Dec. 31. She said it was to be executed in 11,225 m.; it is possible that I had made a mistake, but not at all likely, as I read the suggestion to her with the figures before my eyes. The supposed suggestion of 11,225 m. had been carried out correctly.

I now attempted to find out during hypnosis the patient's mental condition in reference to these suggestions. In reply to my questions she informed me:—(1) That when the suggestions were made in hypnosis she did not calculate when they fell due; (2) That she did not calculate them at any time afterwards during hypnosis. (3) That she had no recollection of them when hypnosis terminated. (4) That no memory of them ever afterwards arose in the waking state. (5) That shortly before their fulfilment she always experienced a motor impulse, *i.e.*, her fingers moved as if to grasp a pencil and to perform the act of writing. (6) That this impulse was immediately followed by the idea of making a cross and writing certain figures. (7) That she never looked at clock or watch until after she had made her record.

Experiments, Wed., Jan. 8, 1896.—No. 10: 4.5 p.m. *Sug.:* in 4,417 m.—No. 11: 4.5 p.m. *Sug.:* in 11,470 m.—No. 12: 4.30 p.m. *Sug.:* in 10,070 m.

As Miss A. stated in hypnosis that she made no calculations, in order to vary the experiments I asked her, as soon as I made the suggestions and before terminating the hypnosis, to calculate when they would fall due and tell me the result. She replied as follows: No. 10, in 3 d. 37 m., or 23 m. to 5 next Saturday afternoon.—No. 11, in 187 h. 50 m., or 7 d. 9 h. 50 m. Next Wednesday morning at 5 m. to 12.—No. 12, in 1,067 h. 40 m., or 6 d. 23 h. and 40 m. 4.20 p m. next Wednesday.

Miss A.'s calculation in No. 10 was 1 h. 5 m. too early. The interval (which was 1 h. too short) having apparently been calculated from 4 o'clock, instead of 4.5 p.m. In No. 11 her calculation was 1 d. 3 h. 20 m. too early. Here (1) 11,270 was taken instead of 11,470, and hence the interval was calculated to be 187 h. 50 m., equalling 7 d. 19 h. 50 m. (2) 7 d. 9 h. 50 m. was given instead of 7 d. 19 h. 50 m. The time falling due was then calculated with this interval (7.19.50), but (3) a mistake of 1 d. was made.

In No. 12 her result was correct, but did not correspond with her calculation. 10,070 m. equals 167 h. 50 m., not 1,067 h. 40 m. Here (1) a cypher was wrongly inserted and (2) 40 m. miscalculated for 50. The latter error was repeated when 6 d. 23 h. 40 m. was given instead of 6 d. 23 h. 50 m.—*Res.:* No. 10, Sat., Jan. 11, 5.42 p.m.: Correct.—No. 11, Thur., Jan. 16, 3.15 p.m.: Correct.—No. 12, Wed., Jan. 15, 4.20 p.m.: Correct.

Remarks.

As the subject had wrongly calculated during hypnosis the time the suggestions fell due, I concluded that she had thus fixed these dates in her own mind and would carry out the experiments in accordance with them. My astonishment was great when they were executed correctly. I re-hypnotised Miss A. and said to her, "You did not carry out these suggestions at the hours you told me they would fall due. Why was this?" She replied, "What I told you was all wrong." "How do you know the other results are right?" "I can't tell you, I only feel that they are." Further questioning elicited no memory of the processes by which the

original mistakes had been corrected. Miss A. assured me that she had never thought of the suggestions from the time they were made; she simply fulfilled them in response to an impulse to write down the figures, and while doing so, neither recalled her calculations nor even the suggestions themselves.

When No. 12 was fulfilled, Miss A. had been hypnotised in my room for an hour and had had no opportunity of consulting the clock. Exactly at 4.20, without waking or opening her eyes, she said she had to make a cross and put down the time—this was preceded by the movement of the fingers already described. From this date I arranged that some of the experiments should fall due when Miss A. visited me, but not, it is important to note, every time she came. They were fulfilled either in the normal waking state or in hypnosis, and Miss A. recorded them herself in my case book. I then at once compared her figures with the actual time and entered the result, this being nearly always witnessed and signed by others. From this date several suggestions were made at each séance. In some cases, the same hour was given as the starting point of all the experiments; in others, varying and even imaginary ones were chosen. In the latter case, the subject was told the actual time, but ordered to carry out the experiment from, say, 2.15 p.m. of the previous day.

Experiments, Wed., Jan. 15, 4.45 p.m.—No. 13, from 4.45 p.m. *Sug.:* in 4,453 m.—No. 14, from 2 p.m. *Sug.:* in 10,470 m.—No. 15, from 2 p.m. *Sug.:* in 10,060 m.

At the time the suggestions were made the patient was again asked in hypnosis to calculate when they would fall due and replied rapidly:—"No. 13, in 722 h. and 33 m., or 11.15 p.m. next Wednesday." "No. 14, in 197 h. and 30 m., or 4.5 p.m. next Wednesday." "No. 15, in 8 d., 5 h. and 30 m., or 4.25 p.m. next Wednesday."

In No. 13, Miss A.'s calculation was 4 d. 4 h. 17 m. too late. 4,453 m. equals 74 h. 13 m., not 722 h. 33 m. Perhaps 4,453 was mistaken for 43,353 equalling 722 h., 33 m. The time of falling due had been calculated from the interval of No. 14.

In No. 14, her calculation was 4 h. 25 m. too early. 10,470 m. equals 174 h. 30 m., not 197 h. 30 m. The time of falling due

was also wrong. There is no explanation for either of these independent errors.

In No. 15, her calculation was 1 h. 45 m. too late. The wrongly calculated interval, 8 d. 5 h. 30 m., corresponded to the wrongly calculated 197 h. 30 m. of No. 14; this interval had apparently remained in the mind. The time of falling due had been calculated with 4.45 as initial time, as in No. 13, instead of 3.0.

Results.—No. 13, Sat., Jan. 18, 6.58 p.m. Correct.—No. 14, Wed., Jan. 22, 8.30 p.m. Correct.—No. 15, Wed., Jan. 22, 2.40 p.m. Correct.

Remarks.—Again the subject's miscalculation did not affect the accuracy of her results, and questioning in hypnosis again failed to revive any memory of the processes by which these had been reached.

Experiments, Wed., Jan. 22, 4.5 p.m.—No. 16. *Sug.:* in 20,180 m.—No. 17. *Sug.:* in 20,160 m.—No. 18. *Sug.:* in 20,140 m. Miss A.'s calculations in hypnosis. "No. 16, in 336 h. 20 m., or 13 d. 20 m. Tues., Feb. 4, at 4.25 p.m." "No. 17, Tues., Feb. 4, at 4.5 p.m." "No. 18, Tues., Feb. 4, at 3.45 p.m." In each instance these calculations were 1 d. too early, but in No. 16, 20, 180 m. was correctly given as 336 h. 20 m.

Results.—No. 16, Feb. 5, 4.25 p.m. Correct.—No. 17, Feb. 5, 4.5 p.m. Correct.—No. 18, Feb. 5, 3.45 p.m. Correct.

Remarks.—On Wed., Feb. 5, I hypnotised Miss A. at 3 p.m. At 3.45, without passing from the hypnotic state, she made a cross and wrote down the correct time. I aroused her at 4 o'clock and she carried out the remaining experiments correctly at 4.5 and at 4.25.

Experiments, Wed., Feb. 5, 4 p.m.—No. 19. *Sug.:* in 10,050 m.—No. 20. *Sug.:* in 10,080 m.—No. 21. *Sug.:* in 10,090 m.—No. 22. *Sug.:* in 840 m.—No. 23. *Sug.:* in 900 m.

Miss A.'s calculations in hypnosis: "No. 19, Wed., Feb. 12, 3.30 p.m." "No. 20, Wed., Feb. 12, 4 p.m." "No. 21, Wed., Feb. 12, 4.10 p.m." "No. 22, Thur., Feb. 6, 6 a.m." "No. 23, Thur., Feb. 6, 7 a.m."

The above, made almost immediately, were correct in every instance.

Results.—No. 19, Wed., Feb. 12, 3.30 p.m. Correct.—No. 20, Wed., Feb. 12, 4 p.m. Correct.—No. 21, Wed., Feb. 12, 4.10 p.m. Correct.—No. 22, Thur., Feb. 6, 6 a.m. Correct.—No. 23 Thur., Feb. 6, 7 a.m. Correct.

Remarks.—When Miss A.'s mother went to her bedroom on

the morning of the 6th, she found her asleep and two pieces of paper on a table by the bedside. On each was a rough cross; on one the figure 6, on the other 7, both very badly written. Miss A. said she had not awakened during the night.

The other suggestions were carried out during hypnosis in my room, the time being marked by Miss A. in my note book and witnessed by others.

The five suggestions were given rapidly one after the other. These, and similar complicated ones, were never read to the subject more than twice, and sometimes only once.

I re-hypnotised Miss A. and questioned her about the suggestions which had been carried out during the night, presumably in natural sleep. She told me she remembered nothing about them, and afterwards, when suggestions were again carried out in natural sleep, her memory was equally at fault.

Experiments, Wed., Feb. 12.—No. 24, 3.30 p.m. *Sug.:* in 2,220 m.—No. 25, 3.30 p.m. *Sug.:* in 2,285 m.—No. 26, 3 p.m. *Sug.:* in 10,115 m.—No. 27, 3 p.m. *Sug.:* in 10,150 m.—No. 28, 4 p.m. *Sug.:* in 20,190 m.

Miss A.'s calculations in hypnosis: "No. 24, in 18 h. and 40 m., or 10.10 to-morrow morning.—No. 25, to-morrow morning at 11.15.—No. 26, next Wednesday at 25 m. to 4 p.m.—No. 27, next Wednesday at 5.30 p.m.—No. 28, a fortnight and half an hour."

The answers to Nos. 26, 27, 28, were given immediately. In No. 24, Miss A.'s calculation was 18 h. and 20 m. too early, but would have been correct if the interval suggested had been 1,120, instead of 2,220 m. In No. 25, her calculation was 18 h. 20 m. too early, but would have been correct had the suggested interval been 1,185, instead of 2,285 m. In No. 26, her calculation was correct. In No. 27, her calculation was 1 h. 20 m. too late. Here the interval seems to have been taken as 7 d. 150 m., instead of 10,150 m. In No. 28, her calculation was correct as far as it went, but the exact time of fulfilment was not given.

At the time I made the suggestions I also calculated when they would fall due, thus: No. 24, Feb. 14, 5 a.m. Wrong; half an hour too late.—No. 25, Feb. 14, 6.5 a.m. Wrong; half an hour too late.—No. 26, Feb. 19, 3.35 p.m. Right.—No. 27, Feb. 19, 4.10 p.m.: Right.—No. 28, Feb. 26, 4.25 p.m. Wrong; 5 m. too soon.

Res.: No. 24, Fri., Feb. 14, 4.30 a.m. Correct.—No. 25, Fri., Feb. 14, 5.35 a.m. Correct.—No. 26, Wed., Feb. 19, 3.35 p.m. Correct.—No. 27, Wed., Feb. 19, 4.10 p.m. Correct. No. 28, Wed., Feb. 26, 4.30 p.m. Correct.

Nos. 24 and 25 were fulfilled during sleep. On the 14th Miss A., on awaking, found papers by her bedside with 4.30 and 5.35 written on them. On the 19th she was hypnotised in my room at 3 p.m., and carried out Nos. 26 and 27 while in hypnosis. On both occasions she wrote the time in my note book, and this was witnessed. I asked her during hypnosis if she remembered my last suggestion (No. 28), made the previous week. She said she did and repeated it correctly, but stated she had never thought of it since, and did not know when it would fall due, or the number of minutes that had elapsed since it was given. She had apparently forgotten that, when the suggestion was given, she had calculated when it would fall due. No. 28 was executed correctly during hypnosis on February 26.

Experiments, Wed., Feb. 19.—No. 29, 3.30 p.m. *Sug.:* in 720 m.—No. 30, 3.30 p.m. *Sug.:* in 780 m.—No. 31, 3.30 p.m. *Sug.:* in 2,160 m.—No. 32, 3 p.m. *Sug.:* in 10,135 m.—No. 33, 3 p.m. *Sug.:* in 20,210 m.

Miss A.'s calculations in hypnosis: These, with the exception of No. 32, were all correct, and her replies were almost instantaneous. No. 32 was said to be due at 2.5 p.m. on Wed., Feb. 26. This was 1 h. 50 m. too early, and represented an interval of 7 d. less 55 m., instead of 7 d. plus 55 m.

Res.—No. 29, Thur., Feb. 20, 3.30 a.m. Correct.—No. 30, Thur., Feb. 20, 4.30 a.m. Correct.—No. 31, Fri., Feb. 21, 3.30 a.m. Correct.—No. 32, Wed., Feb. 26, 3.55 p.m. Correct.—No. 33, Wed., Mar. 4, 3.50 p.m., was written down at 3.48. The calculation, therefore, was correct, but the time appreciation 2 m. too early.

Remarks.—On awaking at 7 o'clock on the morning of the 20th, Miss A. found a piece of paper with 3.30 marked on it, and another with 4.30. On the morning of the 21st she found a piece of paper with 3.30 marked on it. She had no recollection of waking during the night, and, as usual, questioning in hypnosis failed to revive any memory of what she had done. The other suggestions were fulfilled in my room and witnessed by others.

Experiments, Wed., Feb. 26, 3.30 p.m.—No. 34. *Sug.:* in 2,140 m.—No. 35. *Sug.:* in 3,590 m.—No. 36. *Sug.:* in 5,030 m.—No. 37. *Sug.:* in 10,125 m.—No. 38. *Sug.:* in 10,100 m.—No. 39. *Sug.:* in 20,180 m.

Res.: No. 34, Fri., Feb. 28, 3.10 a.m. Correct.—No. 35, Sat., Feb. 29, 3.20 a.m. Correct.—No. 36, Sun., Mar. 1, 3.20 a.m. Correct.—No. 27, due Wed., Mar. 4, at 4.15 p.m., was not

recorded.—No. 38, Wed., Mar. 4, 3.50 p.m., was written down at 3.48. Calculation therefore correct, but time appreciation 2 m. too early.—No. 39, Wed., Mar. 11, 3.50 p.m., was written down at 3.51½. Calculation therefore correct, but time appreciation 1½ m. too late.

Remarks.—These suggestions were only read to Miss A. once; she was then asked to repeat them, and did so correctly, with the exception of No. 37. She was told not to make any calculations. Nos. 34, 35 and 36 were executed during sleep, and the papers, as usual, were found at Miss A.'s bedside in the morning. It is to be noted that 3.50, March 4, the terminal time of No. 38, was also the time at which another suggestion, made a fortnight before, fell due, and which has already been recorded in its proper place. Miss A. stated at 3.48 that she had to make two crosses and to put down 3.50 twice. No. 37, due at 4.15 p.m., Wednesday, March 4, I have no record of. I am not certain whether this was my fault or Miss A.'s; I was hypnotising another patient when the suggestions were fulfilled, and I might well have omitted to enter that one; on the other hand, Miss A. might have failed to carry it out. Three suggestions fell due very quickly, and one of them, as we have seen, belonged to another series. When suggestions were made to fall due in a fortnight and I saw the patient in the week between, I sometimes questioned her in hypnosis as to the unfulfilled ones; she always assured me that she had never thought of them, did not know how much of the time had elapsed, nor when they fell due.

Experiments, Wed., Mar. 4, 3.45 p.m.—No. 40. *Sug.:* in 10,080 m.—No. 41. *Sug.:* in 10,055 m.—No. 42. *Sug.:* in 10,040 m.—No. 43. *Sug.:* in 750 m.—No. 44. *Sug.:* in 2,160 m.—No. 45. *Sug.:* in 2,195 m.—*Res.:* No. 40, Wed., Mar. 11, 3.45 p.m., was written down at 3.44. Calculation correct; time appreciation 1 m. too soon.—No. 41, Wed., Mar. 11, 3.20 p.m., was written down at 3.22. Calculation correct; time appreciation 2 m. too slow.—No. 42, Wed., Mar. 11, 3.5 p.m. Correct.—No. 43, Thur., Mar. 5, 4.15 a.m., during sleep. Correct.—No. 44, Fri., Mar. 6, 3.45 a.m., during sleep. Correct.—No. 45, Fri., Mar. 6, 4.20 a.m., during sleep. Correct.

Remarks.—When these suggestions were given Miss A. was not asked to calculate when they would fall due. Mr. Barkworth and Dr. Barclay were present when Nos. 40, 41 and 42 were fulfilled.

At this séance, March 11, fresh suggestions were made under the following conditions. Mr. Barkworth and Dr. Barclay were both put *en rapport* with Miss A., and it was agreed that they should

each make two time suggestions, arranged so as to fall due at the next séance, when they promised to be present. These were given when I was out of the room, and I was not told what they were until after their fulfilment. The suggestions were as follows :—

Experiments, Wed., Mar. 11, 4 p.m.—No. 46. *Sug.:* in 21,400 m.—No. 47. *Sug.:* in 21,420 m.—No. 48. *Sug.:* in 21,428 m.—No. 29. *Sug.:* in 21,434 m.—*Res.:* No. 46, Thurs., Mar. 26, 12.40 p.m., was written down at 12.38. Calculation correct, time appreciation 2 m. too early.—No. 47, Thur., Mar. 26, 1 p.m., was written down at 12.59. Calculation correct, time appreciation 1 m. too early.—No. 48, Thur., Mar. 26, 1.8 p.m. Correct.—No. 49, Thur. Mar., 26, 1.14 p.m. Correct.

Remarks.—Miss A. was hypnotised at 12.30 p.m. on Thursday, March 26, and carried out the suggestions while in that condition. Mr. Barkworth and Dr. Barclay were both present and checked the time records. None of us, however, had any idea whether the experiments were correctly carried out or not, as Mr. Barkworth and Dr. Barclay had mislaid their notes and were unable to recall the suggestions they had given. Miss A. was roused from the hypnotic state and, as usual, remembered nothing of the suggestions. She was then re-hypnotised, asked to recall them, and replied as follows: "They were made at 4 p.m. last Wednesday week and were to be fulfilled in 21,400, 21,420, 21,428 and 21,434 minutes. Mr. Barkworth and Dr. Barclay gave two suggestions each." Miss A. stated that she had made no calculation at the time and had not thought of the suggestions afterwards. On April 22, Dr. Barclay sent me the lost memorandum of his two suggestions, viz., 21,428 and 21,434 minutes from 4 p.m. on the day already mentioned. On April 27, Mr. Barkworth wrote to tell me that he also had found his lost memorandum, and that the suggestions were 21,400, 21,420, 21,428 and 21,434 minutes, the first two having been made by himself, the two latter by Dr. Barclay. This agreed with Miss A.'s account.

A fresh series of suggestions was made on April 8, some to fall due during the night, others the following week in my presence. The patient lost her papers recording the former, and I was too busy to enter the latter. These are the only experiments in the whole series which are not recorded, and they are omitted for the above reasons. Later, Miss A. found the records of the suggestions, which had been carried out during natural sleep. They were correct.

Experiments, Thur., May 7, 3 p.m.—No. 50. *Sug.:* in 8,650 m.—No. 51. *Sug.:* in 8,680 m.—No. 52. *Sug.:* in 8,700 m.

I still further complicated these by suggesting as follows: "No. 50 is to be fulfilled in the waking state. Five minutes before No. 51 falls due you are to pass into the hypnotic condition. No. 51 is to be fulfilled during hypnosis, but five minutes afterwards you are to pass into the normal waking state and continue in that until after the execution of No. 52. Eight minutes after No. 52 is carried out hypnosis will again appear."

Results.—No. 50. (*a*) Suggestion fulfilled, Wed., May 13, 3.10 p.m. Correct. (*b*) Hypnosis appeared at 3.31 p.m. This ought to have been 3.35 p.m. and was therefore 4 minutes too early.

No. 51, Wed., May 13, 3.40 p.m. (*a*) Suggestion fulfilled during hypnosis. Correct. (*b*) Miss A. passed spontaneously into the normal state at 3.45. Correct.

No. 52, 4 p.m. (*a*) Suggestion fulfilled in the waking state. Correct. (*b*) Hypnosis appeared exactly at 4.8. Correct.

Remarks.—On May 13, Miss A. came into my consulting room at 3.5 p.m., and almost immediately fainted. She had recently met with a severe accident and was in acute suffering. Immediately on regaining consciousness she said she had to make a cross at 3.10, and did so in my case book; others were present in the room when all the suggestions were fulfilled with the exception of the first.

Experiments, Wed., May 13, 4.30 p.m.—The suggestions were given in the following general terms: "You are to repeat all the experiments made last Thursday, but to-day you are to start from 2.55 instead of 3 p.m., and to each suggestion you are to add 1,440 minutes." The original suggestions were not cited, nor any other information given. The experiments, therefore, were as follows:—

No. 53, Wed., May 13, 4.30 p.m. *Sug.:* in 8,650 m. from 3 p.m., plus 1,440 m., minus 5 m. from starting point.—No. 54, Wed., May 13, 4.30 p.m. *Sug.:* in 8,680 m. from 3 p.m., plus 1,440 m., minus 5 m. from starting point.—No. 55, Wed., May 13, 4.30 p.m. *Sug.:* in 8,700 m. plus 1,440 m., minus 5 m. from starting point.

Results.—No. 53, Wed., May 20, 3.5 p.m. Fulfilled in the waking state. Correct. Hypnosis appeared at 3.30. Correct. No. 54, Wed., May 20, 3.35 p.m. In hypnosis. Correct.

Miss A. passed spontaneously into the normal state at 3.40. Correct.

According to the original suggestions, Miss A. was to remain in the normal state until the fulfilment of the next experiment, but, as she had a severe headache, I hypnotised her, made curative suggestions, and told her hypnosis would terminate one minute before the next experiment fell due. She passed into the normal waking state at 3.49, six minutes too soon.—No. 55, Wed., May 20, 3.55 p.m. was written down at 3.50. Calculation, therefore, correct, but time appreciation 5 minutes too early.

I re-hypnotised Miss A. immediately the above experiment was fulfilled. At 4.3 p.m., while still in the hypnotic state, she said it was 3 minutes past 4, and that I had suggested that hypnosis would appear at that hour. This was correct.

Remarks.—It is to be noted that hypnosis appeared at 3.30 p.m., the exact time suggested. This is particularly interesting as the experiment, correctly executed at 3.30 on May 20, was the erroneously carried out experiment of May 13, complicated by five minutes having been deducted from its starting point and 1,440 added to its interval.

No. 55 was the last experiment of the series. A few others, similar in character, were made in October, 1896. These were successful but presented no fresh features, and as Miss A. had to cease her visits, owing to her approaching marriage, further experiment was impossible.

Summary.—Fifty-five experiments are cited; of these one, apparently, was either not carried out by Miss A., or unrecorded by me, while in another (No. 9) she mistook the original suggestion, but fulfilled it correctly in accordance with what she thought it had been. Forty-five were completely successful, *i.e.*, not only did Miss A. write down the correct terminal time, but this was done, also, at the moment the experiment fell due. Eight (Nos. 33, 38, 39, 40, 41, 46, 47, 55) were partially successful. In these the terminal time was correctly recorded in every instance, but there were minute differences, never exceeding five minutes, between the patient's correct estimate of when the suggestion fell due and the moment at which she carried it out. The proportion which these errors bear to their respective intervals varies between 1 to 2,028 and 1 to 21,420. The following table gives an analysis of the conditions under which the experiments were carried out and their results:—

Experiments.		Witnessed by				Results.		Unrecorded	Remarks.
		Friends or relatives.	Bramwell and others.	Bramwell alone.	Unwitnessed.	Correct.	Wrong.		
Fulfilled in the waking state	26	14	11	1	0	20	6	0	1 mistaken suggestion. 1 fulfilled 2 minutes too soon. 1 fulfilled 2 minutes too soon. 1 fulfilled 1 minute too soon. 1 fulfilled 2 minutes too soon. 1 fulfilled 5 minutes too soon.
Fulfilled in hypnosis	15	0	15	0	0	12	3	0	1 fulfilled $1\frac{1}{2}$ minutes too soon. 1 fulfilled 2 minutes too soon. 1 fulfilled 1 minute too soon.
Fulfilled in natural sleep	13	0	0	0	13	13	0	0	In each instance figures which correctly represented the terminal time of the experiment were found at the subject's bedside in the morning, but there is no evidence to show whether this was done at the moment the suggestion fell due, *i.e.*, the subject's calculations were correct, but evidence as to time appreciation is wanting.
Unfulfilled ...	1	0	0	0	1	0	0	1	It is doubtful whether this experiment was carried out by the subject. It is, however, possible that I omitted to record it, as it fell due when I was engaged with another patient.
Total ..	55	14	26	1	14	45	9	1	

Similar experiments, more or less successful, were made with other somnambules, but in none were the results so striking as with Miss A. In those about to be cited the subject was Miss B., aged 20, an intelligent, well-educated girl, who had received some scientific training. Her arithmetical powers were superior to Miss A.'s, but she possessed no particular aptitude for appreciating the passage of time. She was a somnambule, could be rendered anæsthetic and analgesic by suggestion, and had been the subject of several painless minor surgical operations. Her health, from the commencement of the experiments to the present date (August, 1900) has been good.

The first experiments consisted in determining by suggestion the time of waking from normal sleep. The hours selected varied widely; but the results were almost uniformly successful and the greatest error recorded did not exceed five minutes. Others similar to Miss A.'s followed thus:—

Exp. 1, Nov. 25, 1895, 3.55 p.m. *Sug.:* in 24 h. and 50 m. *Res.:* Correct. *Remarks:* In reply to questioning in hypnosis, Miss B. stated that when the suggestion was given she calculated when it would fall due and determined to carry it out at that hour.—*Exp.* 2, Nov. 27, 1.20 p.m. *Sug.:* in 1,445 m. *Res.:* 10 m. too early.—*Exp.* 3, Dec. 6, 3 p.m. *Sug.:* in 1,440 m. *Res.:* Correct.—*Exp.* 4, Dec. 9, 3.15 p.m. *Sug.:* in 2,880 m. *Res.:* Correct.—*Exp.* 5, Dec. 12, 3.30 p.m. *Sug.:* in 1,540 m. *Res.:* 7 m. too late.—*Exp.* 6, Dec. 16, 3.30 p.m. *Sug.:* in 1,620 m. *Res.:* 13 m. too late.—*Exp.* 7, Dec. 20, 3 p.m. *Sug.:* in 1,380 m. *Res.:* Correct.—*Exp.* 8, Dec. 31, 3.15 p.m. *Sug.:* in 24 h. 1,200 m. *Res.:* Correct.—*Exp.* 9, Jan. 2, 1896; 3.10 p.m. *Sug.:* in 24 h. 1,430 m. Miss B.'s calculation, made in hypnosis, was 40 m. too early. *Res.:* 5 m. too late.—*Exp.* 10, Jan. 6, 3.15 p.m. *Sug.:* in 24 h. 100 m. Miss B.'s calculation in hypnosis was correct. *Res.:* 8 m. too late.—*Exp.* 11, Jan. 27, 3.10 p.m. *Sug.:* in 24 h. 150 m. Miss B.'s calculation in hypnosis was correct. *Res.:* 10 m. too soon.—*Exp.* 12, Mar. 27, 3.10 p.m. *Sug.:* in 24 h. 240 m. Miss B.'s calculation in hypnosis was correct. *Res.:* Correct.

Time experiments more or less closely resembling those cited have been occasionally repeated with Miss B. up to the present date (August, 1900) and with practically identical results.

Before considering theoretical explanations of hypnotic and post-hypnotic appreciation of time, I propose to discuss (*A*) the possibilities of mal-observation or deception, and (*B*) to draw attention to certain other points which appear worthy of notice.

(*A*) *The Question of Mal-observation or Deception.*

(1) The subjects of all my time experiments were either former patients or personal friends. None of them were trained hypnotic subjects, and in no single instance was a pecuniary reward promised or given. All this, however, does not in itself exclude the possibility of mal-observation or deception, and I would rather base my arguments in favour of the genuineness of the results on post-hypnotic amnesia, and the fact that the problems involved were beyond the subject's waking powers.

(2) While, however, all observers recognise post-hypnotic amnesia, it must still be admitted that loss of memory might be assumed for purposes of deception. Fortunately, there are other hypnotic phenomena impossible of imitation; amongst these may be cited: (*a*) the absence of certain organic changes following injury (Delbœuf's case of two symmetrical burns), and (*b*) the absence of physiological signs of pain during severe and prolonged operation. The latter fact was clearly demonstrated in the operations on my patients at Goole and Leeds (*Journal of Dental Science*, March 30, 1890, and *Lancet*, April 5, 1890). Several of these patients were afterwards the subjects of my time experiments, and all who were employed for this purpose, including Miss A. and Miss B., could be easily rendered anæsthetic or analgesic by suggestion.

(3) Post-hypnotic amnesia alone, even when it is undoubtedly genuine, does not exclude possible error, as the subject might receive information from the operator or spectators. It is, however, difficult to say how this could have happened in Miss A.'s case. Thus, twenty-seven experiments were fulfilled in my absence, and no information regarding these—excluding, of course, the suggestions

made to Miss A. during hypnosis—was given to anyone until some time after the whole series was completed. I did not calculate when any of these twenty-seven suggestions would fall due, and did not know, until after their fulfilment, whether they had been carried out correctly or not. Twenty-seven further experiments were fulfilled in my presence, these, with one exception, were also witnessed by others. In four of them the suggestions were made by Mr. Barkworth and Dr. Barclay, and I did not know what they were until afterwards. These two operators, however, could not assist the subject, as they had lost the memoranda of their suggestions and were unable to recall the figures. In the remaining twenty-three, none of the spectators knew what the suggestions were. Indeed, in most instances, they did not know that any experiments were being carried on, until they saw them executed and were asked to witness the figures, their ignorance being purposely arranged.

(4) In the twenty-three cases just cited, before giving the suggestions, I calculated when they would fall due. Could Miss A. have learnt anything about this through telepathy or muscle-reading? During the last ten years, I have searched for evidence of telepathy and also taken part in the experiments of other observers; the results, however, have invariably been negative; if, for argument's sake, we conceded the possibility of telepathy, recognising that somnambules possess hyperæsthesia of the special senses, it would still be difficult to see what information Miss A. could have obtained from me. In the majority of the experiments I did not work out when the suggestions would fall due, and even when I did, many of my calculations were only approximately correct, although I was not aware of this until after all the experiments were completed. Moreover, I I have an unusually bad memory for figures, and never either before or during the execution of the suggestions, recalled my calculations as to the time at which they were supposed to fall due. Further, when the experiments were carried out I was nearly always busily engaged with other patients and so placed that Miss A. could not see my face.

(5) Again, even supposing post-hypnotic amnesia had

not existed in Miss A.'s case, the retention in the waking state of the memories of hypnotic life would not in itself explain her feats in calculation and time appreciation. Miss A.'s memory, knowledge of arithmetic and power of appreciating time, in no way exceeded that of other imperfectly educated girls in her station of life. Her normal memory was incapable of retaining complicated series of figures, and she was unable to make even much simpler mental calculations than those involved. After the suggestions were made, she remained in the hypnotic state for an hour or more and could not consult the clock. During this period it was absolutely impossible for her to record the suggestion in any way other than mentally.

(B) Other Points of Interest.

(1) Five minutes before the first experiment was fulfilled, Miss A. became restless and felt she must do something. This preliminary stage of restlessness was absent in all the others. In them, when the time for carrying out the suggestions arrived, Miss A. had a sudden twitching of the fingers of her right hand, immediately followed by the idea of writing down certain figures. The abruptness of this invasion of the normal consciousness, by a message from the subliminal one, was particularly noticeable when Miss A. was actively engaged in conversation at the time.

(2) On twenty-four occasions Miss A. was asked to calculate when the suggestions fell due; she was wrong in the first nine instances, but in the remaining fifteen right in eleven and wrong in four. As the experiments advanced, not only the frequency, but also the extent of Miss A.'s errors in calculation decreased, and the answers were given much more rapidly. Sometimes the correct replies were almost instantaneous, and in these instances no conscious calculation could be traced. It is to be noted that Miss A.'s mistaken calculations had no effect on the correctness of her results.

(3) *Memory.*—Once only did Miss A. spontaneously recall in hypnosis that a time suggestion—yet unfulfilled—had

been given. This was *Experiment No.* 3, where the suggestion was an easily remembered one, viz., 24 hours and 100 minutes. On other occasions when Miss A. was questioned in hypnosis as to the unfulfilled suggestions, she invariably recalled the fact that these had been made, but rarely remembered their exact terms. She always asserted that she had never thought of them, did not know how much time had elapsed since they had been given, nor when they were due. This was so even in cases where she had calculated the terminal time. At first Miss A. forgot all about the suggestions immediately after they were fulfilled; she did not know she had made a cross or written down the figures, and could not recall what they meant. This condition of memory was identical with what is almost universally associated with post-hypnotic acts. Later, for convenience' sake, it was suggested to Miss A. during hypnosis that she should remember having executed the experiments. She then knew in the waking state that she had made a cross and written down certain figures, but recalled nothing of the original suggestion, of which these acts were the fulfilment. When Miss A. was questioned in hypnosis, after the execution of the suggestions, her memory, on certain points, was very clear. She could recall in every detail the terms of all experiments that had recently been carried out, *i.e.*, she remembered the hours at which they had been made, the number of minutes suggested, her own calculations, if any, and the moment and circumstances under which the suggestions had been fulfilled. Putting aside the calculations she made at the time, in response to suggestion, she was unable to recall having made any others, or to give any information as to the methods by means of which she had correctly fulfilled the experiments. When a second series of suggestions was given, before the first had been fulfilled, after all had been carried out, she could recall both series and place each member of them in its proper order. This memory, however, was not persistent. A fortnight after the experiments had been executed, although Miss A. still remembered in hypnosis that they had taken place, she was unable to recall the details. When experi-

ments were fulfilled in normal sleep she remembered their terms in hypnosis and when they had been given, but not when they had been executed.

(4) The experiments had no prejudicial effect on Miss A.'s health. On the contrary, this steadily improved. She is now a strong, healthy, well-developed woman, the mother of two children, and has had no return of her nervous symptoms.

In no single instance did any bad effect, even of the most trivial description, follow hypnotic experiment.

(5) The results of the experiments were only estimated after the series was completed, when a friend, Mr. Bartrum, B.Sc., kindly checked them for me. He discovered that some of my calculations made at the time had been erroneous. I am also indebted to him for a critical examination of the calculations the patient was asked to make when the suggestions were given.

(6) With the following exception, the phenomena observed in the cases of Miss A. and Miss B. differed little. When a simple suggestion was given, Miss B. sometimes spontaneously calculated when it would fall due. Miss A., on the other hand, never made any spontaneous calculations at all. Apparently Miss B. did not spontaneously calculate the more complicated arithmetical problems. When she did so, in response to suggestion, her results were invariably correct, but, despite this, the experiments were not always fulfilled at their appropriate time. Miss A., on the contrary, was often wrong in her calculations, while the suggestions themselves were carried out with phenomenal accuracy.

(7) In some recent instances Miss B. apparently made no spontaneous calculations, despite the fact that the arithmetical problems involved were extremely simple. For example, I suggested that she should shake hands with me forty minutes after I aroused her from hypnosis. At the moment indicated, in the midst of an animated conversation, she suddenly asked me to shake hands with her. In reply to my questions, she said she had felt impelled to do this, but could not tell why. A few minutes later she had entirely forgotten the incident. I re-hypnotised her; she then re-

called the suggestion and the impulse she had experienced, but could not remember having made any calculation or having in any way marked the passage of time.

Theoretical Explanations.

Delbœuf, as we have seen, pointed out that the terminal day in Beaunis and Liégeois' experiments fell on an easily recognised date with which the subjects were acquainted. These experiments, he said, did not show, as they were supposed to do, that somnambules possessed the power of counting days, but only that they were able to retain a given date. To remove this objection his suggestions were made in minutes, while the majority of mine were given in a similar but even more complicated form. In explaining the phenomena, therefore, we must take into account, not only the appreciation of the passage of time, but also the feats of memory and arithmetical calculation which sometimes exceeded the subject's normal powers.

Bernheim's Theory.

This is mainly based on the supposed occurrence of self-hypnosis in somnambules and the existence during that condition of a peculiar mental concentration, with consequent revival of hypnotic memories. According to Bernheim, conscious mental activity exists both in sleep and in hypnosis. During sleep we are conscious that our mind thinks and works, just as the somnambulist knows what he is doing, but the form of consciousness differs from that of waking life. In both, concentration of the nervous force upon the suggested image or idea is the characteristic phenomenon. This continues to exist, although dream succeeds dream in sleep and varying suggestions are instantly executed in hypnosis; the nervous concentration has only changed its object, the focus shifted its place.

According to Bernheim, if an individual goes to sleep determined to wake at a given hour, his attention is fixed

upon this idea, and he thinks about it voluntarily and consciously all night. When he awakes, he believes he has done so spontaneously, as the conscious thoughts of sleep are forgotten. Finally, he says, the lost memories of hypnosis may be revived by chance association of ideas, like forgotten impressions of the waking state, and also in other ways, and thus, the memory of the suggestion has not been latent all the time. In support of this he cites :—

First—*Experiments.*—Two subjects, who had received deferred suggestions, when hypnotised and questioned in the interval, stated that during natural sleep they had once dreamt of what had been suggested during hypnosis.

Secondly—*Certain general observations as to the mental condition in somnambules.*—The memory, he says, of what has taken place during hypnosis depends upon the psychical concentration already referred to; every time this is reproduced, the lost memory is revived. Somnambules pass easily and spontaneously from the normal to the hypnotic state; they then become self-absorbed and concentrated, and recall the operator's suggestions. They know when these should be executed and take their measures accordingly; they reinforce the idea of not forgetting them, just as a person in natural sleep determines not to miss the hour set for waking. Although the suggestion may have been present in their minds the greater part of the day, they forget this if we divert their attention from themselves by speaking to them. By doing this, we have disturbed their psychical concentration, drawn their cerebral activity from the inside to the outside, and produced another state of consciousness in which the memory of the suggestion is lost; the memories of the second, or hypnotic state, being effaced in the first, or normal, one.

In consequence of this, when the somnambule carries out a suggestion, he believes that the idea has newly and spontaneously dawned in his mind—"*He no longer remembers that it is a memory.*" Thus, in Bernheim's opinion, a deferred suggestion is no more difficult to explain than one executed immediately on waking.

Three points are involved in Bernheim's theory.

(1) *Nervous concentration and its relationship to hypnotic memory.*—According to Bernheim, everyone possesses a certain definite amount of nervous force or cerebral activity. In the waking state this is concentrated in the higher nervous centres—the reasoning part of the brain; in hypnosis in the lower ones—the imaginative or automatic part. All the phenomena induced during hypnosis—conceptions, movements, sensations, images—owe their origin to this concentrated and accumulated nervous force. When the subject passes into the normal state this nervous concentration ceases, and the impressions received during hypnosis fade. When hypnosis is again induced, the former state of concentration reappears and, at the same time, the lost memories are revived.

This explanation is ingenious, but not in keeping with observed facts. If I suggest to a subject that he will still remember the events of hypnosis, after that state has terminated, he invariably does so. Yet, according to Bernheim's theory, the redistribution of nervous force to the higher centres should inevitably have prevented this. Again, suggestion may rob the hypnotised subject of the power of recalling the events of previous hypnoses. If, however, Bernheim's explanation be correct, the lost memories could not escape revival, seeing that they must have reappeared when the nervous force was again concentrated in the lower centres.

Further, as numerous and varied hypnotic phenomena can be simultaneously evoked in the same subject, this clearly shows that the phenomena of hypnosis cannot be explained by the concentration of attention on any one given point. Again, the fact that the multiple phenomena are sometimes similar in character to the isolated ones, indicates that the explanation of hypnotic phenomena by means of the amount of attention concentrated is also fallacious. If all the attention is requisite for the production of one hypnotic phenomenon and yet, while it still lasts, many others are simultaneously induced, whence do these derive that excessive amount of attention which is said to be necessary for the induction of the primary phenomenon?

(2) Admitting that hypnotic memory is associated with this psychical concentration, the evidence in favour of the frequent and spontaneous occurrence of the latter is far from convincing. Granting that two subjects had each *once* dreamt of a suggestion during natural sleep, this does not justify the conclusion that somnambules pass easily and spontaneously from the normal to the hypnotic state. Further, although the memories of hypnosis, whether self-induced or not, are lost on waking, these can be easily evoked by questioning in subsequent hypnoses. If, therefore, subjects passed spontaneously into the hypnotic state, and thought about the suggestions, they could be made to recall the fact that they had done so. Despite this, Bernheim's generalisations are founded on the two dreams just cited; and he has apparently made no attempt to discover the actual mental conditions involved, although he might easily have done this by means of the simple method of interrogation in subsequent hypnoses.

(3) Granting both the points in dispute, *i.e.*, the psychical concentration and its spontaneous occurrence, the difficulty is still unsolved. If no trace of the hypnotic memory remains in the normal state, what advantage does the normal consciousness obtain from the unknown or forgotten fact that the hypnotic consciousness recalled the suggestion at some time before the date fixed for its fulfilment?

Beaunis' Objections.

Beaunis also raises many objections to Bernheim's views. Thus, while admitting the influence of attention and concentration upon the production of certain hypnotic phenomena, he holds that these are insufficient to explain deferred suggestions. They may account for a hallucination realised immediately on waking, but not for one that has been retarded for several days. Here, the subject was unconscious of the suggestion until the hallucination appeared at the hour fixed, and it is absurd to suppose that all this time he was under the influence of a dominant idea. Bernheim's experiments, therefore, do not settle the question.

Granting that the subject dreamt of the suggestion in normal sleep two days before it fell due, it is still necessary to explain how it was carried out at the time fixed. The suggestion, it is true, now involves a shorter period of time, *i.e.*, two days instead of the original number, but this, while reducing the extent of the problem, still leaves it unsolved.

Beaunis disputes Bernheim's assertion that those who wish to wake at a given hour think of this all night, for, he says, if this were true, they would afterwards remember having done so. Dreams which have passed rapidly through the brain can often be recalled in every detail; we should, therefore, be able to remember still more vividly the ideas upon which our attention has been continuously fixed. Further, some people always know what o'clock it is, and, if suddenly asked, are able to give an exact reply, no matter how much their attention may have been concentrated on other things. Had they consciously noted the passage of time, they would afterwards be able to recall having done so. This, however, is not the case, and they are unable to explain how the feat was performed.

Finally, in contradistinction to Bernheim, Beaunis justly points out that important differences exist between the way in which the lost memories of the waking state and those of hypnosis are revived. The former may be recalled at any time by a chance association of ideas. For example, things long forgotten may be remembered on hearing the name of someone we knew in childhood. The lost memories of hypnosis, however, possess the distinctive and essential characteristic that they cannot be revived by a chance association of ideas, and are, therefore, fundamentally different from those of the waking state. The hypnotic suggestion is only realised at the hour fixed and cannot take place before, even when associations occur which would have restored lost normal memory. Thus, if it is suggested to A. that he is to do something at the expiration of ten days, when he hears B. cough three times, B.'s signals, if given at any time before the day fixed, produce no response, although they inevitably do so when the appropriate time arrives.

Beaunis' Theory.

Beaunis attempts to explain the phenomena of time appreciation by the existence in the human organism of an unconscious physiological power of time measurement. The phenomena of deferred suggestion, he says, is analogous to that of awaking at a fixed hour from normal sleep, and both may be explained by the existence of a sort of mental mechanism, arranged, like an alarm clock, to produce a movement at a fixed hour. The brain is a machine which acts without our knowledge, with an activity we are unable to estimate, and the things of which we are conscious only feebly represent this mysterious work. The power of appreciating time, rudimentary or rather atrophied in civilised man, is well developed in the savage and lower animals. Thus a dog, accustomed to go out with his master at a certain hour, will show that the time has arrived by expressive pantomime, should his master delay a little in getting ready for his walk. In Beaunis' opinion, the measurement of time by somnambules is an act of unconscious cerebration.

Paul Janet's Objections.

Janet grants that an image of which one is unconscious may exist in the memory, and, further, that it can be revived, even at a fixed date, if the operator associates the suggestion with some definite sensation, as, for example, the sight of a particular person. He cannot, however, understand the return of the lost memory at a fixed day, without other association than the numeration of time. Thirteen days, for example, do not represent a sensation, but form an abstraction. The carrying out of a suggestion, therefore, at the expiration of such a period, presupposes the existence of an unconscious power of measuring time—an entirely unknown faculty. Up to this everything could be explained by the law of association of ideas, but here we make a sudden jump, and the thread of analogy is completely broken. No association can explain the unconscious counting of thirteen days, and the "suggestion theory" is in default.

Beaunis' Reply.

A day is not an abstraction. The idea of a day represents a series of definite impressions, the result of external agencies, such as light, temperature, &c., which produce in our organism different kinds of reactions. A day is, then in reality, not a sensation, but a succession of sensations and unconscious reactions. Animals know exactly the hour at which they are habitually fed, and an attack of fever returns at the same hour each day, or every other day, for weeks; this indicates that the measurement of time is not an abstraction, but has its roots in the very life of the organism. The periodicity of days, weeks, months and seasons corresponds to periodic organic variations, which under certain circumstances might acquire sufficient intensity to constitute a sort of "unconscious faculty for measuring time," although the word "faculty" is a little too philosophic a term for an organic aptitude of this kind.

Remarks.

Beaunis' explanation assumes (1) the existence of an unconscious faculty for measuring time, and (2) its applicability to the cases in question.

(1) *The unconscious measurement of time.*—According to Beaunis, time appreciation is highly developed in savages, while certain civilised persons can awake at a given hour, and others always know what o'clock it is. In support of this, however, he does not cite a single example or experiment. Let us take the simplest case, *i.e.*, waking at a given hour. Many persons, it is true, believe they can do this; usually, however, the self-waker only shows the power of giving himself a lighter sleep than usual; he wakes several times during the night, and, in the end, does not always hit the exact hour. On behalf of the Society for Psychical Research, several persons, who believed they possessed this power of self-waking, tested it in a rigorously scientific manner. The results were disappointing. One experimenter only succeeded on two occasions out of seventeen, and one of these was his usual hour of waking; another was successful on five times out of twenty-nine, but on three of these he

awoke at other hours as well, while the third was right two out of thirteen times, and the fourth succeeded in four instances out of forty-six.

Since I became interested in time appreciation, many people have assured me that they could awake at any given hour. None of them, however, were able to experimentally reproduce the alleged phenomenon, while the two following are the only genuine cases I know of :—

Dr. George Savage possesses the power of waking at a given hour and has tested it on many occasions. The following is an example :—One day, having to catch an early train, he determined to wake at 6 a.m., and slept soundly without waking until the exact time. The seven following mornings he woke exactly at six, notwithstanding that he went to bed at different hours, and there was no necessity for early rising. This involuntary repetition of self-waking at unusual times also occurred when he was roused by others at abnormally early hours. Thus, when in the Alps, if he were called at 2 or 3 a.m., he would certainly wake spontaneously at the same hour next morning, even if he had been much fatigued with climbing. Dr. Savage states that the accuracy of the time of waking in these instances has puzzled him greatly.

The following is Professor Marcus Hartog's own account of his case :—

"When I was a student, under 17, I found I could, sleeping soundly, awake at any given time I had set myself overnight. The peculiarity of such waking was that it was always sudden and complete, not preceded by a period of light broken sleep, nor accompanied by the drowsiness of an ordinary unprepared waking. If I found that it was needless to get up I soon fell asleep again, and then had the ordinary drowsy waking, often oversleeping myself. This faculty has persisted with me. Between the ages of 20 and 25, on three distinct occasions I had to nurse friends, when I had to administer food and medicines at regular intervals, as well as to attend to every call or unrest on the part of the patient. The last occasion extended over, I think, three weeks. On each occasion the facility and manner of waking completely and suddenly was exactly the same as for early rising, whether at the stated call, or at the least stir of the patient. On lying down to rest again I seemed to see a gradually widening vista, and as my eyes diverged I fell asleep; the time

occupied could not have been a quarter of a minute, though I felt wide awake at the moment of closing my eyes. My sleep on these occasions was singularly if not absolutely dreamless, though I was under the greatest mental anxiety while waking."

In Professor Hartog's case the waking at fixed hours was never involuntarily repeated, *i.e.*, as regards the waking at repeated intervals. Sometimes, however, when he set himself to wake at a fixed time in the morning this was repeated for several days, as in Dr. Savage's case.

(2) *The applicability of Beaunis' theory to the particular cases in question.*—The experiences of Savage and Hartog undoubtedly show that certain persons can wake at a given hour. This does not, however, explain why this power is almost universal amongst somnambules; it is an attempt to explain the little known in terms of the less known, and the analogy, even if successfully established, does not solve the problem but adds another to it. Granting that the uncivilised man possesses some natural power of marking the passage of time, it would be of little use to him in cases such as those cited by Delbœuf and myself. If he cannot count above five how is he to bring his powers to bear on a suggestion which starts from, say, 3.15 p.m. yesterday and is to terminate in 20,840 minutes?

Delbœuf's Theory.

Delbœuf first pointed out that his subjects in the normal state were unable to make the necessary calculations involved in his time experiments. As the latter were successful this showed, he said, that the subjects, when hypnotised, had an idea of the passage of time and unconsciously calculated when the suggestions fell due. Apparently they possessed a sort of mechanism like an alarm which they set to go off at a fixed time.

Edmund Gurney's Objections to the Theories of Delbœuf and Beaunis.

According to Gurney, commands to be fulfilled at a particular date involved the reckoning of time. This was

usually assumed to be physiological. Thus, Delbœuf believed that the subject calculated when the order fell due, and said to himself: "I shall fall into a trance at such and such a moment and then perform the act." The subject set his organism like an alarm for a given time ahead; his mind, relieved of responsibility, then went off duty till the suggestion fell due, when it was aroused by its own automatic machinery, the action resembling the running down of an alarm. Gurney asserted that hypnotic subjects never formulated their orders in this way, and that Delbœuf's explanation was pure guess work, unsupported by fact.

Further, he said, even if we granted the purely physiological hypothesis for cases of short duration, where the idea of the time fixed could be easily grasped, and thus the setter of the alarm knew exactly what he was about—it would be a very different thing to extend it to others where the command had been executed after the lapse of months.

Gurney, like Delbœuf, drew a distinction between commands to be carried out at a specific date, such as New Year's Day, and those where the length of time alone was named, as in the direction to do something "on the 69th day from this." In the former instance the brain might at once register the date along with the order, and the arrival of the former would thus suffice to arouse the latter. A length of time could not be registered in this way. Its termination, till reckoned by the calendar, was quite indefinite, and when the particular day arrived it conveyed nothing likely to revive the suggestion—it carried no more *sixty-ninthness* about it than any other day.

Gurney considered that Beaunis' explanation did not fully meet Janet's objection that such a length of time—*i.e.*, 69 days—was simply an abstraction. Admitting that "a day" was a sufficiently familiar and definite unit to present a concrete character, that it represented a series of conscious reactions, and, further, that there were periodic organic changes which extended over weeks and months, this did not prove that "the measurement of time was not an abstraction, but had its roots and conditions in the very

life of the organism." It did not follow that "sixty-nine days" were concrete in character because this was the case with "one day," and, further, the organic conditions associated with established physiological periods were absent from those suddenly and arbitrarily fixed by human volition. The vital processes were as unable to make a time calculation of this kind as a school boy's digestion to work out a proposition of Euclid. Such time measurement was not a function of animal life; its result was not an inevitable bodily state, but a needless act. It depended—not on progressive changes in the stomach or blood—but on an original course of cerebration taking place in the higher tracts of the brain, initiated by an impression—that of the command—which had a distinct psychical side.

According to Gurney, the passage of time must be registered, looking at it from the brain side alone, not by general gradual change, but by a series of specific changes corresponding to the days and units of measurement, and this was the only kind of cerebral process capable of clearly differentiating the case from that of ordinary physiological time reckoning. Further, unless cerebral events such as were normally correlated with the ideas "sixty," "sixty-one," "sixty-two," &c., really took place, how could the gulf be spanned with precision? Any other kind of change would not know when to stop, or how to associate a point it had reached with the order given long before. Granting that these specific brain-changes took place, was it not reasonable to suppose that their mental correlate existed; and that, hidden from our view, there was an actual watching of the course of time? This hypothesis went a long way towards removing Paul Janet's difficulty. From this point of view his "unknown faculty" resolved itself into a known one, working in the normal way, but below the surface of ordinary consciousness.

Gurney's Theory.

As we have seen, the carrying out of a post-hypnotic command several thousand minutes after waking can neither be explained by any ordinary physiological power of appre-

ciating time, nor by a supposed spontaneous re-hypnosis with temporary revival of memory. The feat, according to Gurney, can only be performed as the result of the intelligent action of a secondary consciousness, which watches the time as it passes just as the ordinary one would do, and, when the correct hour arrives, pushes the ordinary consciousness on one side, takes its place and carries out the command. In Gurney's opinion, the mental conditions involved in the execution of post-hynotic commands varied widely in different cases. Of these the following are examples:—

(1) A subject was told to do something ten minutes after she awoke. On waking she looked at the clock until the expiration of the time, then executed the order. In this case, the subject was conscious of having consulted the clock, and afterwards—apart from any question of the revival of hypnotic memory in post-hypnotic states—remembered having done so.

(2) A subject was told to do something five minutes after he awoke. Immediately on waking he looked at the clock and continued to do so at intervals, talking naturally meanwhile to those present. At the end of five minutes he executed the order. Afterwards, he neither recollected looking at the clock, nor carrying out the command.

This case appears to occupy an intermediate position between No. 1 and the others about to be cited.

(3) A subject was told that on the 39th day from then he was to execute a post-hypnotic suggestion. He had no memory of the command when awake, and no reference was made to it until March 19, when he was suddenly asked, during hypnosis, how many days had elapsed since it was given. He instantly replied, "Sixteen," and added that there were twenty-three more to run. Both statements were correct.

(4) Another subject was told on March 26, that he was to do something on the 123rd day from then. On April 18, he was hypnotised and asked if he remembered the order. He at once replied: "Yes, this is the twenty-third day; a hundred more." Further questioning made it clear that every few days the command occurred to his mind and that

he calculated how many days had passed and how many more had to elapse. His waking memory retained no recollection of the original suggestion, nor was it aware of the memories and calculations which he described when hypnotised.

In the two last cases, the watching was undoubtedly wholly of an internal kind, and, although obviously accompanied by consciousness, was afterwards entirely forgotten.

The two points in Gurney's theory which specially demand consideration are: (I.) What evidence have we of the existence of a secondary consciousness? (II.) Granting its existence, how far does it explain all the phenomena we have been considering?

(I.) *The secondary or subliminal consciousness.*

The facts in favour of the existence of a secondary consciousness may be divided into three groups: (*a*) Those arising from, or more or less closely associated with, morbid states; (*b*) Those occurring in normal waking or sleeping life; (*c*) Those arising spontaneously, or as the result of suggestion, in the hypnotic condition.

(*a*) To those who have not studied hypnotic and cognate phenomena, this group is practically the only one that exists. Its most familiar examples are to be found in post-epileptic and other more or less clearly defined morbid conditions. Here the personality of the secondary consciousness is generally a reduced one; volition is frequently suspended or weakened, and consciousness dimmed. The individual does not recognise himself, his surroundings and mental state, in as clear a fashion as he would do were he in his normal condition.

The case of Louis V. is one of the most interesting of this kind. This young man presented two distinct mental, moral, and physical states. In the primary, he was docile and intelligent, while sensibility and movement were normal. In the secondary, he was violent, vicious, less intelligent, and presented hysterical contractures. Each state was completely independent of the other and possessed absolutely distinct memories.—"The Life History of a Case of Double or Multiple Personality," by A. T. Myers, M.D. (*Journal of Medical Science*, January, 1896); "Les Variations de la personnalité," par MM. Bourru et Burot (*Revue de l'Hypnotisme*, 1887, p. 193).

Dr. Rieger (*Der Hypnotismus*, 1884, pp. 109-115) cites a case of frequently recurring attacks of secondary consciousness in an epileptic. In the normal condition his character was orderly, but this state alternated with others during which he would leave his home for weeks at a time and lead the life of a thief and vagabond, sometimes being sent to prison, sometimes to asylums. In his primary condition, he had no memory of the acts for which he had been punished.

Dr. Lewis C. Bruce (BRAIN) gives an account of an asylum patient who not only showed two separate and distinct states of consciousness, but in whom the right and left brain alternately exerted a preponderating influence over the motor functions. At one time he was ambidextrous and only understood English, at another he was left-handed and spoke Welsh.

Spontaneous somnambulism affords another example of the action of a secondary consciousness. In volition and consciousness are to be found the main differences between it and induced somnambulism. The hypnotic subject knows what he is doing and why he does it, and can reject disagreeable suggestions. Unless specially trained, he usually only acts in response to influence from without—influence more or less directly transmitted to him by the suggestions of others. The spontaneous somnambule, on the contrary, acts his own dreams. His hallucinations sometimes excite criminal acts, and authentic cases are reported in which spontaneous somnambules have committed murder, under the delusion that the person they attacked was a wild beast or a burglar. Spontaneous somnambulism, properly speaking, is a neurosis, and, as Hack Tuke pointed out, in its severest forms approaches perilously near epilepsy, while the condition of mental automatism which sometimes succeeds epileptic fits closely resembles that of the sleep-walker. Yellowlees looks upon spontaneous somnambulism as a form of insanity, and calls it *somnomania*. He holds that the sole difference between it and insanity or epileptic violence is that it occurs during sleep, and involves only a temporary arrest of volition, instead of the more prolonged loss of control which results from organic mischief.

In another group of cases the secondary state, while markedly inferior to that particular individual's primary one,

finds its counterpart in degenerate states which are not necessarily insane.

Of this the following case, recorded by "Dagonet" (G. R. Sims) in the *Referee*, January 31, 1897, is an example:—One night Mr. Sims received a message from the master of a workhouse, to the effect that the police had brought a man to the infirmary whom they had found insensible in the street, apparently suffering from drink; a letter, signed by Mr. Sims, was in his pocket. Mr. Sims knew him as a professional man in a good position. Next day he was conscious, but absolutely denied his identity. He said he was a street musician, and that his cornet had been stolen during a row in a public house where he was playing. He returned to his lodgings as soon as he was discharged from the infirmary, and told his landlady he thought he must have had a fit and fallen down in the street, for he was bruised and ached all over, but did not remember anything about it. Exclusive of the time passed in the infirmary, he had been absent from his lodgings a week, but maintained that he had only left the house that morning. A few days later he fulfilled an important professional engagement. All went well for some time, then he again suddenly disappeared. Search was made for him, and he was eventually found playing a cornet outside a public house in Camden Town. Addressed by his proper name he made no sign, and when questioned asserted that he had been an itinerant musician for the last fifteen years. To-day Mr. —— is again living an ordinary respectable professional life, and has not the slightest knowledge of his periodical lapses into another individuality—that of a street musician. His name is well known to all who are familiar with music and the drama.

In another group, the secondary state, instead of being one of obvious mental disorder, differs little in intelligence and volition from the primary one. The following are examples:—

Ansel Bourne, an itinerant preacher, aged 61, residing at Greene, Rhode Island, suddenly disappeared. In spite of the publicity which the newspapers gave to the fact, and the efforts of the police to find him, he remained undiscovered for two months. He then found himself at Norristown, Pennsylvania, where for the previous six weeks he had been keeping a small variety store under the name of A. J. Brown, appearing to his neighbours and customers as a normal person. When his normal

consciousness returned, he was extremely alarmed to find himself in an unknown situation, with absolutely no memory of his surroundings, or of the incidents which had taken place from the date of his disappearance.—*Proceedings of the Society for Psychical Research*, 1891, p. 221.

Emile X., barrister, sometimes passed, without loss of consciousness, into a secondary condition, which lasted from a few minutes to several days. During it he led an active life, which apparently did not differ from his normal one. The state always terminated suddenly, and the ordinary consciousness retained no recollection of what had just passed. After one attack, which lasted three weeks, he came to himself a hundred miles from home.—Dr. Proust, Professor of Hygiene at the Hôtel Dieu, Paris. *Revue de l'Hypnotisme*, March, 1890.

Mr. N., aged 32, educated, consulted Forel and told him the following story. He stated that he had been living for some weeks in Zürich, and had vague recollections of arriving there after a long voyage. Recently he had read two newspaper notices describing the sudden disappearance from Australia of a Mr. N., and he felt that he was the person referred to, although at the same time it seemed impossible that this could be true. N. entered the Burghölzli Asylum for treatment and was hypnotised by Forel, who succeeded in restoring his lost memory by suggestion, and thus obtained an account of his doings from the time he had left Australia. There was no reason, pecuniary or moral, for N.'s disappearance. He recovered under Forel's treatment.—*Ein Fall von temporäner, totaler, theilweise retrograder Amnesie (durch Suggestion geheilt)*, von M. Naef, Voluntärarzt an der Heilanstalt Burghölzli. *Zeitschrift für Hypnotismus*, 1897, p. 321.

The following is the most recent example I have been able to trace of the group we are considering:—On April 16, 1900, George Ridderband, aged 19, a law student and the son of a well-known lawyer, suddenly disappeared from his home in New York. No reason, except that he had been studying hard for an examination, was discovered for this, either at the time or afterwards. His parents put the matter in the hands of the police. On April 21 the lad walked into a police station and told the following story:—Five days previously, he suddenly found himself walking in the street, but neither knew who, nor where he was, nor could he recall anything as to his past life. Since then he had been wandering about, struggling in vain to revive his lost memories, keeping meanwhile a diary of his proceedings. He asked if any-

one corresponding to him had been advertised as missing, and the sergeant recognised him as the lost George Ridderband. He was taken home and placed under medical care. At first he did not recognise any of his relatives, but a week later his memory commenced to return; he was still, however, unable to account in any way for his disappearance.

In another group the secondary state, from a physical, moral and intellectual point of view, is superior to that of the primary one. The following are examples:—

A girl, aged 18, healthy, but quiet and somewhat stupid in character, swallowed some unguentum lyttæ. After the acute symptoms of poisoning passed off, a cutaneous hyperæsthesia of the head and morbid sensitiveness to sound remained. She had frequent attacks of temporary insensibility and passed alternately from her ordinary mental condition to a secondary one, and from this in its turn back again to the normal one. In the secondary state she was excitable; her conversation was lively and spirited, and she was intellectually superior to what she was in the primary condition. The memories of the two states were absolutely distinct; the primary self knew nothing of the secondary one, the secondary self nothing of the primary one.—"Case of Double Consciousness," by Thomas Mayo, M.D., F.R.C.S., *London Medical Gazette*, New Series, Vol. I., 1845, p. 1202.

Mary Reynolds, a dull and melancholy young woman, was found one morning in a profound sleep, from which it was impossible to arouse her. After twenty hours she awoke, but her disposition was absolutely changed and she had no memory of her past. She was now cheerful, buoyant, sociable and merry, but did not recognise her relatives or friends, and had lost the power of reading and writing. Five weeks later, she passed into the primary state; again became dull and melancholy, and remembered nothing of her secondary condition. After the lapse of a few more weeks she again passed into the secondary stage after a profound sleep, and took up her new life precisely where she had left it. The memories of the two states were absolutely distinct; in the first she remembered former primary states, but these alone, in the second, she only remembered former secondary states. These alternations of consciousness continued for fifteen years, then finally ceased, leaving her permanently in the second state. In this she remained without change for the last twenty-five years of her life.—"Case of Mary Reynolds," by Dr. Weir Mitchell. *Trans. of the College of Physicians of Philadelphia*, April 4, 1888.

Félida X., aged 14½, without known cause, though sometimes under the influence of emotion, had attacks of sharp pain in both temples, followed by a state of profound stupor, which lasted ten minutes. She then spontaneously opened her eyes and appeared to wake, but in reality passed into a condition of secondary consciousness. This lasted for an hour or two, then the stupor and sleep re-appeared and she passed into her ordinary state. The secondary state differed markedly from the primary one. In the latter she was a miserable, querulous, hysterical invalid, and remembered nothing of her secondary life, which was superior, both intellectually and physically, to her primary one. In it she was gay, active and intelligent, and remembered not only all the events which had taken place in former attacks of secondary consciousness, but also those of normal life. As time went on the frequency of the secondary attacks became greater and their duration longer, till at the age of 24 they commenced to exceed the periods of normal life. From 24 to 27 she remained in the normal state. After this the secondary attacks became more and more frequent, and finally almost completely occupied her entire existence. In 1875 Félida, who was then 32 years of age, told Azam that she still suffered from attacks associated with loss of memory. These so-called "attacks," however, were simply lapses from her secondary consciousness into her original primary one. Thus, once when returning from a funeral she felt her attack, *i.e.*, her normal state, come on. She became unconscious for a few seconds, without her companions noticing it, then woke in the primary state, absolutely ignorant of the reason for which she was in a mourning carriage. Accustomed to these accidents she waited till, by skilful questions, she was able to grasp the situation, and thus none of those present knew what had happened. Later, she lost her sister-in-law after a long illness, and, during a relapse into the normal state, knew nothing about her death, and only guessed at it from the fact that she was in mourning. In the earlier periods the transition from one state to another was marked by a period of more or less prolonged unconsciousness. As time went on, this diminished and, finally, the loss of consciousness became so brief that Félida was able to disguise it. In 1887, when Azam published the account of the case, Félida was 44 years of age and her lapses into normal life had become more and more rare.—*Hypnotisme double Conscience et Altérations de la Personnalité*, par le Dr. Azam. Paris, 1887, J. B. Ballière et Fils.

In 1888 Dr. Ladame, of Geneva, communicated a somewhat

analogous case to the Société Médico-Psychologique, which had been cured by suggestion.

In the cases just cited, the first attack of secondary consciousness was frequently preceded by shock, although sometimes it arose without apparent exciting cause. In subsequent ones it was usually more difficult to trace the exciting cause; generally the attacks were independent of the patient's volition, although cases are recorded by McNish and others where the state could be induced at will.

The attacks of secondary consciousness usually terminated spontaneously, and without discoverable cause. In some instances, however, the accidental reading of an account of their lost selves seems to have evoked some of the memories of the primary state and contributed to the disappearance of the secondary one.

(*b*) *Facts in support of a secondary consciousness drawn from normal waking or sleeping life.*—Here the evidence is not so clear as in the preceding section, but without the hypothesis of a secondary consciousness, it is difficult to explain phenomena such as the following:—

(1) Time appreciation in cases such as those of Savage and Hartog. Here, no ordinary physiological explanation is possible, and, while the ordinary consciousness was asleep, an intelligence of some kind must have watched the passage of time.

(2) The recollection of forgotten facts without the association of ideas connected with normal memory. Everyone, I think, must recall instances in which he has tried to remember a forgotten name, and, after having failed to recall it by every device imaginable, at last put the problem on one side in disgust and turned his attention to other things. Later, the forgotten name rises into consciousness, with a suddenness which is startling, and no association of ideas can be discovered to account for its appearance.

(3) Literary problems, which are vainly struggled with when awake, are sometimes apparently solved in sleep. Thus, on awaking, page after page is easily and satisfactorily

written, while the day before the author had been unable to find expression for his thoughts.

(4) According to Myers, works of genius, instead of being the result of an infinite capacity for taking trouble, are due to the intelligent action of a secondary consciousness. The labour is performed in a subterranean workshop, as it were, and then presented in completed form to the normal consciousness. The latter not only believes that it has done the work itself, but thinks that this has been performed instantaneously.

(*c*) *Hypnotic phenomena which apparently support the theory of a secondary consciousness.*—These are too numerous to be referred to in detail, and it might even be claimed that every deeply hypnotised subject shows evidence—in memory changes alone—of the existence of a secondary consciousness. In the waking state, he can recall nothing of what has passed in profound hypnosis, but when hypnotised, not only remembers the events of previous hypnoses, but also all that his ordinary memory can recall of waking life and even, in addition, much that it had forgotten. Further, he has acquired a control over his own mind and body without parallel in waking life. He can alter the rhythm of his pulse, control his secretions and excretions, and increase or arrest the activity of his special senses. He can induce anæsthesia and analgesia, and, even when all the phenomena are elicited by the suggestion of the operator who has hypnotised him, maintain consciousness and volition unimpaired. From the therapeutic side he can obtain relief from the pain of disease or injury, procure sleep at will and for as long or as short a time as he wishes. He can escape from obsessions, conquer the diseased craving for alcohol and narcotics, and get rid of numerous functional nervous disturbances. Finally, he can be taught to hypnotise himself, and from henceforth—free from all external interference—can by self-suggestion produce phenomena identical with those just described. All these phenomena cannot be evoked in every case, but something can always be effected beyond the power of the waking will.

The following is an interesting example of the spontaneous

and intelligent action of the secondary consciousness in an untrained hypnotic subject. I questioned Miss A. (the subject of the time experiments above referred to) during hypnosis, as to her mental life in previous hypnoses. She replied as follows:—"When you do not speak to me and nothing occurs that interests me directly, I generally think of nothing and pass into a condition of profound restfulness. Once, however, I had an important dress to make and was puzzled how to do it. After you had hypnotised me and left me resting quietly, I planned the dress. When I awoke, I did not know I had done so and was still troubled about it. On my way home, I suddenly thought how the dress ought to be made and afterwards successfully carried out my ideas. I believed I had found the way out of the difficulty there and then in the waking state, I now know I did so some hours previously when hypnotised." When the subject was aroused, she had no recollection of what she had said and still believed she had planned the dress when awake. The origin of this feat in dressmaking—one apparently beyond the subject's normal powers—may be taken as illustrating the possible source of the higher intellectual achievements of genius.

Finally, there is evidence which shows, not only that there is a secondary consciousness which alternates with the primary one, but also that the two may act simultaneously and independently of each other. This is clearly demonstrated by the phenomenon of automatic writing.

Automatic Writing.

In choosing a subject for automatic writing two things are essential, viz.: (1) he must be a somnambule, *i.e.*, retain no memory on waking of what has passed in the hypnotic state, and (2) hypnosis must be capable of being induced and terminated instantaneously. Thus, when the subject is aroused immediately after the suggestion has been given, the problem must be solved in the waking state, although, as we shall see, not by the waking consciousness.

I have often made the following and similar experiments.

I ask a subject while awake to write down a few verses; these I take charge of and do not show him again. I then make him read aloud from some book previously unknown to him, this being chosen in order to engage his entire attention. While reading I hypnotise him suddenly, place pencil and paper near his right hand, and suggest: "On waking you will go on reading where you left off, and at the same time write down how often *b* (or any other letter selected) occurs in the verses you gave me. Wake up." He wakes, resumes reading, and at the same time writes down the answer to the problem suggested. This, almost invariably correct, is often done so rapidly that I have not had time to count the letters, even with the verses before me. I now tell the subject to stop reading, and ask him what he has written. He replies, "Nothing," and when I show him the paper, is astonished and declares he does not know what it means. I then re-hypnotise him, whereupon the lost memory returns, and he not only recalls the suggestion, but also the fact that he has carried it out.

Thus, the primary waking consciousness retains no recollection of the hypnotic suggestions. It does not know that the secondary consciousness, after the hypnotic state has been terminated, first solves the problems, and then directs the motor acts which record them. It is also unconscious of the motor acts themselves.

Gurney made many interesting experiments with healthy non-hysterical men, which illustrate the severance of the normal, or primary, from the latent, or secondary, consciousness. Of these the following are examples:—(1) The first were simple cases which involved memory, but not independent thought. Thus, Gurney showed P., one of his subjects, a planchette and made him write his name with it. P. was then hypnotised; told that it had been as dark as night in London on the previous day, and that he would record this fact in writing. On awaking he remembered nothing. His hand was then placed on the planchette—a large screen being held in front of his face, so that it was impossible for him to see the paper or the instrument—and in less than a minute he wrote, "It was a dark day in

London yesterday." (2) In the next experiments statements were impressed on the subjects, but nothing was said as to subsequently recording them. After waking, however, the writing was executed as before. (3) Gurney made more complicated experiments with another subject. During hypnosis questions were asked about his past life, or arithmetical problems suggested. He was then awakened immediately, before he had time to think of a reply, and, to engross his attention, told to count backwards from a hundred; meanwhile the planchette wrote the correct solutions of the problems. (4) Further experiments involved the reckoning of time. These, however, were not confined to the execution of an order at a given moment, but involved, in addition, other calculations made in the waking state *at a suddenly selected moment*, regarding which nothing had been previously said to the subject. For instance, during hypnosis, he was told to do something at a given date, and also that, before this time arrived, he would be required to write down the number of minutes that had passed since the suggestion was given, as well as the number that had to elapse before its fulfilment. In the interval, when his hand was placed upon the planchette, he generally wrote the answers to the problems. The results, allowing for the time occupied in writing, were remarkably accurate.

Remarks.

The experiments in groups Nos. 3 and 4 showed, according to Gurney, that the hypnotic substrata included higher physical functions than mere random spurts of memory, viz., processes of deliberate reckoning and reflection, which it would be almost impossible to conceive as having only a physiological existence. In group 4, the order itself was remembered and realised by the secondary consciousness, while the primary consciousness was wholly without knowledge of it. In Gurney's opinion, these so-called automatic writings were, in most cases at all events, intelligent and involved mentation. When a statement was made which the subject was told he was afterwards to record in writing, the performance might possibly be regarded as an exclusively

automatic one, due to a "setting of the organism." Sometimes, however, the impression was made without a hint as to the future—without the faintest suggestion that it would produce any result whatever; yet, in the midst of quite irrelevant surroundings and experiences, the phenomenon appeared as soon as the opportunity of "automatic" representation arose. Here the organism could not have been specially set for the effect. Further, if we regarded the action as purely automatic, we should be compelled to concede a singular power to hypnotic impressions, viz., that of storing up energy in the brain which would work mechanically outwards along the motor nerves, as soon as the act of writing was sufficiently easy for the muscles. Moreover, memory on re-hypnotisation afforded strong evidence of mentation. Not only did the subject remember the original idea conveyed to him, and the fact that he had written something, but also the exact words he had used to convey his conception of the impression. Surely this indicated an intelligent apprehension of the words. We have seen that the memory of these so-called automatic acts could be recalled in hypnosis, but some of them, in addition, indicated the existence of a hypnotic memory superior to the normal one, as the facts recorded had occurred at an early period of the subject's life and had long been forgotten.

(II.) *Granting the existence of a secondary consciousness, how far does this explain all the phenomena we have been considering?*

The facts just cited—and these might be supplemented by many more — apparently indicate not only that a secondary consciousness exists, but also that it can act simultaneously with, and independently of, the primary one. Even, however, if we grant the existence of a secondary consciousness, this does not explain all the phenomena of time appreciation and arithmetical calculation that we have been considering.

Obviously if the intelligence of the secondary consciousness were at all comparable to that of the primary one, it would not be difficult for it to calculate the arrival of the

39th or the 123rd day from any given date. In Gurney's cases, it apparently performed this feat with ease, and the subject, when hypnotised, could recall the manner in which it had been accomplished.

Here the secondary consciousness apparently acted in just the same way as the primary one might have done under similar circumstances; having to execute an order on the 39th day from a given date, it recalled the fact from time to time and noted how many days had passed and how many had to come. Many of the experiments I have cited, notably the more complicated ones executed by Miss A., present more difficult problems, and by none of the theories already considered can these be properly solved. Bernheim's explanation, as we have seen, is in opposition to observed facts, while Gurney's objections to that of Beaunis apply with even greater force to my cases. Thus, if there is little analogy between ordinary physiological periods of time, and, for example, 39 days chosen suddenly and arbitrarily by human volition, the analogy is still less between the former and, say, 20,845 minutes.

Further, the secondary consciousness theory, while it affords a reasonable explanation of the way in which Gurney's subjects carried out their suggestions, fails to account for much that is important in the cases I have cited. The following are the principal points which demand explanation:—(1) The want of hypnotic memory as to the manner in which the suggestions were carried out. (2) The fact that the suggestions not only involved feats of arithmetical calculation and memory far beyond the subject's normal powers, but also in some cases beyond their hypnotic ones. (3) The difference in the nature of the time appreciation required in Gurney's cases and in those cited by me.

(1) *The want of hypnotic memory as to the manner in which the suggestions were carried out.*

According to Bernheim, the hypnotic subject is conscious in all stages, and all the memories of hypnotic life, which are lost on waking, can be restored by suggestion or other means. This statement is not absolutely correct. Hypnotic life, it is true, is more conscious than the normal one,

and lacks the prolonged and regularly recurring periods of unconsciousness, represented in the latter by sleep. Sometimes, however, deeply hypnotised subjects receive impressions, and even perform acts, of which they are unconscious.

Thus, (*a*) where anæsthesia has been induced for surgical purposes, the subjects can recall nothing of the operations, either when awake or in subsequent hypnoses. Generally speaking, present forgetfulness does not prove past unconsciousness. When we find, however—in the instances where analgesia and hyperæsthesia have been simultaneously excited—amnesia as to certain sensations, associated with hyperæsthesia as to others, we have reasonable grounds for inferring that the sensations, which cannot be recalled, never reached consciousness. This view is further strengthened by the fact that the operations were characterised by absence of shock, persistence of analgesia after waking and unusual rapidity in the healing process.

(*b*) If a subject is told to be unconscious of everything until aroused by the operator, he will, if neither questioned nor touched, take no notice of what is said or done around him, while suggestion, in subsequent hypnoses, often fails to revive any memory of what has taken place.

(*c*) If a simple movement is suggested, of which volition does not disapprove, it may after a time become automatic, *i.e.*, after having been frequently consciously and voluntarily performed, it may be executed unconsciously, as a genuine automatic act, in response to the habitual stimulus which has excited it. Here, again, the lost memory cannot be recalled by suggestion in subsequent hypnoses.

With the exception of examples of the three classes just cited, the acts of hypnotic life are performed consciously and can be recalled by suggestion in subsequent hypnoses. Further, as the hypnotic memory is more exact and far-reaching than the normal one, the absence of memory as to some given circumstances of hypnotic life, other than those just cited, is markedly suspicious. These suspicions are deepened when the forgotten act is analogous to those invariably remembered by the hypnotised subject. Gurney's subjects easily remembered their calculations as to the days

that had passed, those that had to come and the terminal time of the experiment. In some instances, when the arithmetical problems involved were simple, Miss B., in subsequent hypnosis, recalled the fact that she had calculated out the terminal time of the suggestions and set herself to carry it out at the appointed hour. This, by the way, Gurney asserted hypnotised subjects never did. When the experiments became more complicated, Miss B. ceased to do this, and then, like Miss A., was unable to revive in hypnosis the slightest trace of memory, as to the manner in which the suggestions had been carried out—they could then recall no calculations and no time watching, no foretelling of the terminal time and no recognition of it when it arrived. Yet, from what we know of hypnotic memory, it is impossible to doubt that if calculations and observations of this kind had been made by the subject, they would have been able to have remembered them when again hypnotised.

(2) *The fact that the suggestions not only involved feats of arithmetical calculation and memory beyond the subject's normal powers, but also in some cases beyond their hypnotic ones.*

Delbœuf pointed out that his subjects, in the normal state, were unable to make the necessary calculations involved in his time experiments; therefore the success of these experiments, he said, demonstrated that the subjects when hypnotised had an idea of the passage of time, and unconsciously calculated when the suggestions fell due. Experiments made in hypnosis, however, showed that the subject was quite incapable of calculating even much simpler problems than those involved, and, although this fact was recorded by Delbœuf himself, he apparently missed its significance. When Miss A. was asked to calculate in hypnosis when the suggestions fell due, she was wrong in the first nine instances, her errors frequently being extremely gross ones. Again, unless specially asked to do so, the secondary consciousness apparently made no calculations at all. The time appreciation, however, could not be carried out independently of *such* calculations, obviously the subject could not perform an act at the expiration of 40,845 minutes

unless she—or some self, or consciousness, within her—knew the terminal time this represented.

Further, in Miss A.'s case the hypnotic memory was apparently incapable of retaining the complicated series of figures which were read to her. When she was questioned about them in subsequent hypnosis, but before the fulfilment of the suggestions, she always recalled that the latter had been made, but rarely correctly remembered their details —her recollection being less and less distinct in proportion to the time which had elapsed since they had been received.

(3) *The difference in the nature of the time appreciation required in Gurney's case and in those cited by me.*

There is a marked difference between the recognition of a particular day on its arrival and the last minute in such a series as 20,845. A secondary self or intelligence, which can count the days that have passed and those that have to come and refreshes its memory by doing this every night, could have no difficulty in recognising the terminal day. The varying impressions from the external world, which tell us that a new day has dawned, would be received as freely by the secondary as by the primary consciousness, and all that the former would have to do would be to associate them with the calculation it had made the night before, that the next day would be the suggested one. In my cases the problem is a widely different one. Granting that some intelligence worked out the arithmetical portion, and determined that the suggestion fell due, for example, at 3.25 p.m. a fortnight later, the determination of the arrival of 3.25 differs widely from the recognition of the dawning of a particular day. Admitting that some intelligence, equal to the primary one, tried to determine when this moment arrived, circumstances, often specially arranged, added to the difficulties of the task. In some instances, for example, Miss A. was in a darkened room for several hours before the suggestion was executed, and absolutely without any of the ordinary methods of determining the time. Even if she knew what o'clock it was when she entered the room at noon, how could she determine when it was 3.25?

The general conditions of memory, then, in reference to the experiments I have cited, apparently show that the secondary consciousness—or at all events such manifestations of it as Gurney describes—did not participate in them at all. Further, the feats in calculation and memory were beyond the power of that consciousness, while we have no evidence of the secondary consciousness carrying out time appreciations comparable to the particular cases involved. Some intelligence, however, must have made the arithmetical calculations, and, further, corrected them when they were erroneously worked out by the secondary consciousness. Something also must have remembered the complicated series of figures and the varying results of the calculations, and also, in some fashion, noticed the time as it passed and connected this with the date given as the result of the calculations involved in the suggestions. Could the problems have been worked out by a third consciousness acting independently of the primary one? The theory that a multiple, as well as a secondary, consciousness exists is not a new one, although, as far as I know, it has not yet done duty in explaining the phenomena of time appreciation. I propose now to examine some of the alleged cases of multiple consciousness, and later to see what light, if any, they throw upon the problems we have been considering.

Cases of So-called Multiple Consciousness.

Sarah L. was the first person in whom I found any evidence of so-called multiple personalities, and when she came under my notice about ten years ago I knew nothing of the literature on the subject. In the normal state she was quiet, respectful and somewhat shy, and retained this character when I hypnotised her. She was a profound somnambule; in the waking life she knew nothing of the events of hypnosis, but in hypnosis could recall all the memories of the normal state as well as the incidents of hypnotic life. In 1889, the late Mr. Bendelack Hewetson, of Leeds, operated on her for double strabismus, the only anæsthetic employed being hypnotic suggestion. Later, she sustained a

severe fracture of the nose; there was a good deal of displacement and the parts were swollen and extremely painful. Profound anæsthesia was again induced by suggestion and the bones moulded into position; in each instance the occurrence of subsequent pain was entirely prevented by suggestion. Further, the patient was also cured of attacks of migraine, from which she had suffered since the age of 7, while, after fifteen months' amenorrhœa, menstruation appeared at a suggested date. The above details are cited in order to show the genuine and profound character of the hypnosis.

Before coming under my care Sarah L. had been hypnotised and exhibited by a stage performer, and, some time after the events just recorded, her mother told me that Sarah occasionally hypnotised herself, and that the condition then differed markedly from the one I induced. After some coaxing the girl consented to hypnotise herself, and went through the following performance:—First, she closed her eyes and appeared to pass into a lethargic state, then, a few minutes later, awoke with a changed expression; instead of having a shy and modest air, her eyes sparkled and she looked full of mischief. In place of addressing me as "Sir" —she had formerly been a servant of mine, and an extremely respectful one—she put her hand on my arm and said in a familiar way, "I say?" She then began to ask me impertinent questions about the persons she had seen at my house, and to criticise them in a particularly free and sarcastic fashion. The performance was so interesting and amusing that I got the patient to hypnotise herself on a good many occasions. The same phenomena always appeared; she invariably became familiar, inquisitive and sarcastic, while her highest praise for anyone she approved of was conveyed in the words, "She'll do." Other facts were noticed and reported to me by her mother. Thus, sometimes when Sarah hypnotised herself, she would remember that her mother owed her a few coppers and insist upon having them at once. If they were refused, she would take them by force; to this, however, her mother lent herself. The girl would then generally go out and buy oranges, and,

on her return, eat them all herself; this selfishness was quite out of keeping with her normal character. Sometimes, when her mother asked her to do work that was distasteful to her, such as blacking the grate, she would induce self-hypnosis with profound lethargy and remain apparently deeply asleep for hours. I never taught her to hypnotise herself, nor, as far as I could learn, had this been done by anyone else. During this third stage she remembered everything that had happened in previous conditions of this kind, and also the events of the normal state and those of ordinary hypnosis, but the waking consciousness and the ordinary hypnotic one knew nothing of the events of this third state. After I had observed the condition for some time, I concluded that it was not likely to be beneficial to the patient, and suggested, during ordinary hypnosis, that she should lose the power of creating it. The suggestion was successful and the condition never reappeared.

Pierre Janet has been fortunate in finding several patients who apparently possess multiplex personalities, and has published full and interesting accounts of their cases. Amongst these that of Léonie —— is, I think, one of the most striking. This patient, aged 45, apparently possessed three distinct and well-marked personalities, viz., Léonie I.; the Léonie of normal life, a serious and sad peasant woman, calm, slow, gentle and timid. Léonie II.; Léonie in the ordinary hypnotic state, a gay, noisy, restless being, given to irony and bitter jesting, who describes her visitors in an impertinent fashion and apes their airs and graces. Léonie II. refuses to identify herself with Léonie I., and calls the latter a stupid woman. Léonie has had attacks of natural somnambulism since she was three years old, and from 16 upwards has been frequently hypnotised by various people. Now, when Léonie II. is called to the front, she knits together the events of her natural and induced hypnotic state and forms the history of her life from them; the memories of waking life are not forgotten, but these are ascribed to Léonie I.

When Léonie II. is hypnotised more deeply there appears a third personality, Léonie III., who is grave, serious,

slow in speech and movement. This Léonie separates herself both from Léonie I. of waking life and the Léonie II. of the ordinary hypnotic state. The former she describes as a good, but rather stupid woman, and the latter as crazy. "Fortunately," she says, "there is nothing of me in either of them." Léonie I. knows herself alone; Léonie II. herself and Léonie I., while Léonie III. not only knows herself but also the two others. Léonie I. has only a visual consciousness; Léonie II. has both visual and auditory, while Léonie III. has a visual, auditory and tactile one. Léonie I. is embarassed and ashamed when Léonie II.'s friends, who are strangers to her, speak to her in the street. Léonie II. spontaneously writes letters, which Léonie I. finds and destroys, as she does not understand them. Afterwards Léonie II. hides her letters where she knows Léonie I. will never look for them. Léonie II. visits places where Léonie I. has never been, then disappears and pushes Léonie I. to the front, leaving her frightened by her strange surroundings.

According to Janet these three personalities, which deny and despise each other, not only existed in successive forms, but did so simultaneously. While Léonie I., for example, is apparently the only Léonie, other personalities are inside of the same woman, each of them sensible and wide awake and occupied with her own affairs.

In "An Experimental Study of Visions" (BRAIN, 1898) Dr. Morton Prince records some interesting experiments which he made with Miss X., a patient who suffered from hysterical neurasthenia. Miss X. was easily hypnotised and at first passed into the ordinary deep stage, then, when ordered to sleep more profoundly, a fresh condition developed, which differed both from that of waking life and ordinary hypnosis. According to Dr. Prince those stages formed three distinct personalities, viz.:—

X. I. The Miss X. of ordinary waking life, who is reserved, morbidly conscientious, self-contained, serious, deferential and dignified.

X. II. The Miss X. of the primary hypnotic condition, who is sad, serious, and apparently weak and suffering.

X. III. The Miss X. of the secondary or deeper hypnotic

state, who is flippant and jovial, free from all physical infirmities, full of fun and reckless.

X. I. remembers the events of waking life alone and knows nothing of X. II. and X. III., while X. II. remembers all that has passed in previous primary hypnotic states, also all that X. I. can recall, and, in addition, some other events of waking life which X. I. has forgotten.

X. III. remembers all the events of the secondary or deeper hypnotic stages, as well as everything X. I. and X. II. can recall. In addition, she can describe incidents in the past life of X. I. which are lost to the memory of the latter, and can thus explain much that the waking personality is at a loss to account for. She knows all about many of the little absent-minded doings of X. I., and does not hesitate to voluntarily tell of them, although X. I. is morbidly and unnecessarily reserved about her whole life.

Dr. Prince made this tripartite personage look into a globe, which took the place of the ordinary glass ball used in crystal gazing, and describes the visions which the different personages saw. The following is one of the most interesting of these :—

X. III. was the person in action and voluntarily related the following incident, telling it with great gusto as a joke against Miss X. (X. I.), whom she talked of as "She." "'She' received a letter from a photographer yesterday. 'She' put it into her pocket where 'She' had some bank-notes. Then, as 'She' walked along, 'She' took out the money and tore it to pieces, thinking it was the letter. 'She' threw the money into the street." In response to questioning X. III. repeated the words of the photographer's note and counted mentally, with some difficulty and concentration of thought, the amount of money. X. III. said that "She" was absent-minded and thinking of something else when "She" tore up the money. Hypnosis was now terminated and the ordinary personality, X. I., appeared, who, in reply to questions, stated that she had received a letter from her photographer which she had torn up, and that she had the bank-notes in her pocket. She was asked to show them and produced the letter. This surprised her,

but she thought she must have left the bank-notes at home, and could not believe she had destroyed them. The notes were undoubtedly lost, and an account of the occurrence, similar to that given by X. III., was obtained by making Miss X. (X. I.) look into the globe and suggesting that she should see what had really happened. Here, apparently, the ordinary hypnotic state, namely, X. II., was induced by indirect suggestion and fixed gazing, and the vision appeared in response to the suggestions of the operator.

Crystal gazing, the method employed by Dr. Prince, is not really necessary for the production of hypnotic visions; it simply acts by presenting a point upon which the suggested memories are concentrated, and rendered, as it were, objective. Any memory which exists in the hypnotic consciousness can be evoked by suggestion without the crystal. Under ordinary circumstances the hypnotic subject rarely visualises his memories, but when he looks into the crystal this is a direct suggestion that he should see with his eyes what has happened. I have frequently made similar experiments with hypnotic subjects, and, in place of the crystal, made them look at the top of my stethoscope, which answered equally well.

The following account is condensed from the notes kindly supplied me by Dr. Albert Wilson, of Leytonstone.

Mary W., born October, 1882, had in April, 1895, an attack of meningitis associated with influenza. During the third and fourth weeks of the illness there was high temperature, with delirium bordering on mania, and she called people snakes and did not recognise her friends. In the fifth week, during convalescence, her character changed, and she began to give those around her names which were not their own—thus, her father was Tom, her mother Mary Ann, &c. About the sixth week attacks of secondary personality appeared. The patient would suddenly turn a somersault on the bed and then assume a new character—returning suddenly to the normal, and resuming what she had been occupied with before the attack. At first the seizures lasted from ten to fifty minutes, but increased to hours, days and weeks as the time went on. The secondary self knew

nothing of the primary one and *vice versâ;* further, the secondary self had apparently lost much of the knowledge the primary one had acquired. Thus, in the second state, the patient did not know what her legs and arms were and was childish in her talk. She could write her name, however, but this she did backwards, beginning at the tail of the last letter and writing quickly from right to left—not mirror writing.

After a few months the periods of normal life become shorter, and in place of one secondary stage various others showed themselves from time to time. Of these there were sixteen in all, termed by Dr. Wilson stages *a*, *b*, *c*, *d* . . . *p*. In each stage the subject remembered what had happened during previous attacks of the same stage, but knew nothing of what had occurred in any of the other stages, while the primary personality remembered events in the normal life alone, and knew nothing regarding any of the incidents which had happened in any of the numerous other states.

At the end of a year the normal condition rarely appeared, and then only as a flash—sometimes coming to the surface for five or ten minutes, sometimes only for a few seconds.

The following stages or personalities were noted, most of them being named by the patient herself:—

(*a*) The patient calls herself "Thing"; she is vacant, knows nothing of her past life and cannot stand.

(*b*) Calls herself "Old Nick" and is passionate and mischievous.

(*c*) Here there is catalepsy with deaf-mutism, but the patient writes down all she wants.

(*d*) In this stage the patient has forgotten not only the incidents of normal life, but apparently also much of the general knowledge acquired during it. In writing she spells backwards in the manner already described.

(*e*) A stage characterised by terror.

(*f*) Here the patient calls herself "Good Thing," and is docile, but usually without power in feet or hands; this stage, however, is quite distinct from (*a*).

(*g*) The patient now calls herself "Pretty dear"; is sweet and amiable, but cannot write or spell.

(*h*) Calls herself "Mamie Wud"; in this stage she recalls the events of childhood better than when awake, but is unable to remember anything about her illness.

(*i*) This stage is somewhat like (*d*), when in it the patient knows nothing and thinks she is just born.

(*k*) Here the patient calls herself "Old Persuader," and asks for a stick to strike people with if they won't do what she wishes.

(*l*) She now calls herself "Tom's darling," and is apparently a nice child.

(*m*) In this stage she asserts that she has "no name," and is violent and unkind.

(*n*) She now calls herself "The dreadful wicked thing"; throws her slippers into the fire in a temper, &c.

(*o*) In this stage she calls herself "Tommy's lamb," and is blind and idiotic.

(*p*) In December, 1896, a further stage developed, which lasted till February, 1897. The patient now kept saying "picters," and drew beautifully, even being able to do so when she was prevented from seeing the paper on which she was drawing. The original self was unable to draw. During this stage the pupils were dilated and reacted feebly to light, while the patient was apparently insensible to sound.

As time went on, the patient, when in abnormal stages, began to have some idea of what had taken place in her ordinary life; when in the latter condition, however, she still had no recollection of anything that had happened in any of the abnormal stages. In July, 1898, she passed into a condition much more closely resembling the normal one; she was still, however, childish and called herself "Critter Wood."

At the present time (Summer, 1900) five years since the commencement of her illness, she has apparently settled down in stage (*g*), but has been taught that Mary W., not "Pretty dear," is her name. She is a fine, healthy, well-developed girl, who helps in the house and is anxious to learn typewriting in order to keep herself. Her character, however, differs slightly from her original one, and she is still somewhat childish at times.

Gurney stated that he had experimentally demonstrated the existence of positive and distinct stages of memory within the conscious portion of the hypnotic trance. The subjects of these experiments were first hypnotised lightly, and something was told them which they were asked to remember. Deeper hypnosis was then induced, when the subject was asked what he had just been told; it was found that he neither remembered what had been related to him, nor even the fact that he had been told anything at all. While in this deep stage, which Gurney termed *B*, some new incident was related to the subject which he was again asked to remember. He was then recalled to the lighter stage, termed *A* by Gurney, and asked to repeat what he had been told; he had forgotten what he had heard a few minutes before in stage *B*, but repeated instead what had been told him in the earlier stage *A*, in which he now again found himself. Brought once more to *B*, he similarly remembered what he had been told in that state, while he was again completely oblivious of what had been impressed upon him in *A*. On waking he retained no memory of anything that had been told him in either stage.

Many cases, of which the following is an example, were cited by Gurney to illustrate these alterations in hypnotic memory:—

S., a young man living at Brighton, was told in state *A*, the lighter stage of hypnotic trance, that the pier head had been washed away, and in state *B*, the deeper stage of trance, that an engine boiler had burst at Brighton station and killed several people. He was then brought from stage *B* back again to *A*, when he recalled all that had been told him about the accident to the pier. Hypnosis was again deepened; stage *B* appeared, and the following conversation took place between the operator and the subject:—

Operator: "But I suppose they will soon be able to build a new one."

Had the pier been present in S.'s mind, this remark, said Gurney, would naturally have been taken to refer to it, as it had formed the subject of conversation only a few seconds

before. S., however, at once replied: "Oh, there are plenty on the line"—meaning plenty of engines.

Operator: "The pile-driving takes time, though."

S.: "Pile-driving? Well, I don't know anything about engines myself."

The subject was now brought back to stage *A*, and the conversation continued:—

Operator: "If they have plenty more, it does not matter much."

S.: "Oh, they can't put it on in a day; it was a splendid place."

Operator: "Why, I am talking about the engine."

S.: "Engine! What, on the pier? I never noticed one there."

Here the subject's mind had evidently again reverted to what had been told him in stage *A*.

In the above case, as in all others of a like nature cited by Gurney, the normal self knew nothing of stages *A* and *B*, and these knew nothing of each other.

Before considering multiple personality, in reference to feats of calculation and time appreciation, I wish to examine the characteristics of the various examples of secondary and multiple personality we have been considering. The cases, which present many different phenomena, may be grouped as follows:—

Class I.—Those more or less closely associated with morbid states, and presenting phenomena which range from amnesiæ with alterations in character, to conditions similar to those presented by Félida X.

Class II.—Cases of ordinary hypnotic somnambulism, which afford evidence of a secondary personality.

Class III.—Cases of hypnotic somnambulism, presumably showing evidence of multiple personality.

Class IV.—Time appreciation, &c., like those of George Savage and Marcus Hartog.

Class V.—Instances where mental work, varying from the recollection of a forgotten name to the so-called inspirations of genius, have apparently been carried out by some consciousness other than the ordinary one, and in some condition other than hypnosis.

Class I.

At the lower end of this class Hyslop, in his article on "Double Consciousness," *British Medical Journal*, September 23, 1899, includes cases in which the patients, after a seizure of some sort, performed certain acts of which their ordinary consciousness was ignorant. These varied from purposeless automatic ones to thefts committed with more or less skill or cunning, and, apparently, were neither recalled in the normal state nor in subsequent attacks. At the other end of the group Hyslop cites Félida X., whose case, already reported, differs in many respects from those just referred to. The changes in memory, whether modifications or disturbances, are the most important phenomena in connection with double and multiple personalities, and in Class I. these may be grouped as follows:—

(*a*) Cases where purposeless automatic acts occur, regarding which there is no subsequent revival of memory in any normal or abnormal state.

(*b*) Where more or less intelligent acts are executed, which again are never subsequently recalled, either in primary or in secondary states.

(*c*) Here there are two or more conditions, each characterised by distinct chains of memory: (1) The primary or normal, in which the events of ordinary life alone are remembered, and (2) the secondary or multiple, in which the occurrences of these states are alone recalled.

(*d*) Here the primary state only possesses a memory of its own life, while the secondary has a memory of its own life, and that of the primary one also. Further, the secondary self can often recall much which the primary has forgotten.

Many of the cases also differ widely in their moral and physical aspect. Thus some of the patients, who were gentle and good in the normal state, became violent and criminal in the secondary one. Others changed in the reverse fashion both morally and physically; some, like Félida, who were ill-tempered, querulous invalids in their ordinary condition, became bright, cheerful, good-natured, and more intelligent in their secondary one.

CLASS II.—*Cases of ordinary hypnotic double personality.*

These correspond with the subdivision (*d*) just given under Class I., and the secondary self is always richer in memories than the primary one. It is also, generally speaking, healthier and more refined.

CLASS III.—*Cases of hypnotic somnambulism, presumably associated with multiplex personality.*

Here varying and important changes in memory are to be observed. In typical cases we have (*a*) a primary condition, which knows itself alone; (*b*) a secondary one, which knows itself and the primary one; and (*c*) a third, which knows itself and the other two also, and can recall, in addition, much which has been unconscious in the primary life, or that has been forgotten by the primary consciousness. Usually, the secondary self is superior to the primary one, morally, intellectually, and physically, while the third, in its turn, rises above the second. Sometimes, however, as in Dr. Prince's case, the third personality appears like an evil sprite, who rejoices at the misfortunes of the primary one.

In some cases, such as those cited by Gurney, the two hypnotic selves had each a separate chain of memory and held, as it were, no communication with each other.

In Miss A.'s case the third personality only showed itself in its results—calculations, time appreciation, &c., and I never succeeded in artificially tapping it for purposes of examination. Had I been able to do so, doubtless I should have found that it could recall the calculations, &c., which presumably it had made. It is to be noted that the secondary consciousness was incapable of performing the feats in arithmetic, &c., which the tertiary one found no difficulty in executing.

CLASS IV.—*Time Appreciations, like those of George Savage and Marcus Hartog.*

Neither of these observers can trace any memory of the process by which they arrived at their results. This, how-

ever, does not prove unconsciousness or forgetfulness, as far as some personality is concerned, it only shows that, as in Miss A.'s case, we have not discovered the way of evoking the memories. The repetition of Savage's early wakings on subsequent unnecessary occasions, apparently showed that he was capable of starting, but not of regulating, the mechanism on which these depended, while Hartog apparently possessed more control over his secondary self.

CLASS V.—*Cases of Subconscious Performance of Mental Work in Non-hypnotic Conditions.*

Here, again, it has been impossible to revive any memory as to the means by which the work has been done. Judging from the analogies of hypnotic life—we may instance particularly the dressmaking experience already referred to—it seems probable that such memories exist, and that the secondary self knows how and when it did the work, which the primary one flatters itself it has performed.

The importance of memory in relation to subconscious states is insisted on, both by William James and Myers. According to the former, the theory of "double consciousness" is only, after all, a development of what is found in Locke's famous chapter on "Identity and Diversity," namely, that personality extended no further than consciousness and that there would be two different persons in one man, if the experiences undergone by that man fell into two groups, each gathered into a distinct focus of recollection. Further, says James, "It must be admitted that, in certain persons at least, the total possible consciousnesses may be split into parts which coexist, but mutually ignore each other and share the objects of knowledge between them, and—more remarkable still—are complementary."

In Myers' opinion the formation of a secondary chain of memory is the fundamental point in hypnosis and not mere susceptibility to hypnosis. This chain of memory is essential to the grouping of the various acts which manifest the secondary consciousness. This classification excludes, and I think justly so, some of the cases cited by Hyslop.

Amnesia, even when associated with alterations in character, does not constitute double consciousness. If it did the bather who had forgotten the number of his machine and lost his temper, might be described as a case of secondary personality.

What are the exciting causes of the secondary and multiple personalities?

To this question no satisfactory answer has been given. Shock, hysteria, &c., are said to be the origin of the phenomena in Class I., but these terms explain nothing. We know little of the essential conditions associated with these morbid states, nor why they should produce a severing of the personality in some instances and not in others.

Hypnotic conditions are induced by certain well-recognised methods, but I can see no logical connection between the acts of fixed gazing, concentration of attention, suggested ideas of drowsy states, and the widely varying phenomena of hypnosis. The latter do not appear spontaneously, and some of the methods described must have been employed in every case before primary hypnosis was induced, but I cannot conceive the idea that the methods explain the phenomena.

At first, I considered cases of so-called multiple personalities—where these were hypnotic in origin—as simple subdivisions of the ordinary hypnotic state, produced artificially by suggestion. The appearance of the phenomena of appreciation and calculation of time in absolutely untrained subjects—phenomena which the secondary consciousness could not produce and of whose workings it was ignorant—has caused me to modify this view.

The time appreciation of Savage and Hartog and the so-called inspirations of genius—the results of mental work, which come suddenly, as a finished product, into the primary consciousness—are as difficult to explain, as the hypnotic cases we have just been considering.

Multiple personalities, in relation to time appreciation and calculation.

In the experiments cited, especially Miss A.'s, we noticed (1) that the ordinary hypnotic consciousness possessed no

recollection of having made the calculations necessary for finding out the terminal time, and (2) could not recall any form of time-watching. Further (3), the ordinary hypnotic memory was incapable of retaining the complicated series of suggestions; it remembered they had been made, but could not recall their details. The results of the time appreciation and calculation, presumably due to a third consciousness, appeared as a sudden uprush into whatever personality—waking, sleeping, or ordinary hypnotic—happened to be present at the moment. When the ordinary hypnotic consciousness had been asked to make the necessary calculations and did so erroneously, the third personality ignored these mistakes and did its work correctly. Further, the passage of the message from the third to the second personality, seemed to make a particularly strong impression upon the latter, or to leave, for some time at all events, the door of communication open between the two. Thus the second personality, which had forgotten the details of the suggestions before these were carried out, could recall them accurately a fortnight after they had been executed.

Granting that the third consciousness or personality performed the feats in calculation and time appreciation, the question still remains: "How was it able to do it?" It matters little whether we regard the third personality as really distinct, or as an artificial division of the secondary one, we have still to solve the problems as to the nature and origin of the powers of the hypnotic self, and particularly their capacity for dealing with time appreciations and calculations, such as we have been considering.

Theories as to the Origin of the Powers, or Phenomena, of the Secondary and Multiple Selves.

Delbœuf's Theory.—According to Delboeuf, the various animal functions which the human waking will is now unable to influence, but which the hypnotic self can control, were the revival of a power possessed by lower animal forms —the remote ancestors of our race. These powers had dropped out of the ordinary consciousness in the process of

evolution, as their association with it had become unnecessary in the struggle for existence. Granting that a limited analogy exists between lower animal types and hypnotised subjects, as to their power of influencing certain physical conditions, it would, I think, be impossible to establish an analogy between the mental and moral capacities of the latter and those of the savage or lower animal. The arithmetical powers suddenly developed by Miss A. are not likely to have been derived from some savage ancestor, who was unable to count beyond five, or from some lower animal presumably ignorant of arithmetic. Again, the same subject spontaneously solved, in hypnosis, a difficult problem in dressmaking. The ability to correctly design a garment, in accordance with the present fashion of the day, can hardly have been derived from some woad-stained ancestor, or lower animal form.

Hyslop's Theory.—Hyslop ("Double Consciousness," *British Medical Journal*, September 23, 1899), attempts to explain the phenomena of double consciousness, by a supposed *inhibition of the amœboid movements in the pseudopodic protoplasmic prolongations of the neurospongium.* As the result of this, the connection between the nerve-cells is interrupted, and the nerve currents which, under ordinary circumstances, would pass freely from one to another, now cease to do so. These physiological phenomena, he says, have their psychical counterpart; the interruption of the nerve currents, on the physical side, being represented, on the mental one, by a breaking of the seriality of thought and a loss of the elements, which go to make up consciousness and consciousness of self. To this theory various objections might be raised:—(1) The conditions which Hyslop would explain by it—"the amnesic defects which," he says, "go so far to constitute what we call double consciousness" are just those conditions which are not characteristic of secondary consciousness. It is true that some of the cases he cites, as illustrating double consciousness, were practically characterised by amnesia alone, but these, as I pointed out, must be excluded from the category, as they lack that chain of memory which is essential to the manifestation of double

consciousness. Félida X., also cited by Hyslop as an example of double consciousness, must be taken as the characteristic and fully developed type of the condition we are discussing. Here, the secondary self was not only superior physically and mentally to the primary one, but it also far surpassed it in memory. As time went on, the primary self only appeared as occasional attacks of amnesia, and thus, if it is essential to have a theory to explain amnesic defects, it is those of the primary, and not of the secondary self, which demand solution. Further, those very amnesiæ of the primary consciousness form a part of the memories of the secondary one. In Félida's case the secondary personality ultimately gained a complete ascendancy over the primary one, and, after a time, her periods of normal life—rare of occurrence and short in duration—appeared to her only as an illness, characterised by forgetfulness of events occurring in what was now, to her, her normal existence. In this secondary state, there was neither decrease of volition nor anything wanting of that complete self which forms consciousness.

Hyslop, it is true, confines himself to instances of double consciousness, associated with morbid conditions, but the phenomena presented by Félida X., notably those of memory, are closely analogous to those characteristic of the hypnotic state, and his theory, if correct, should be equally applicable to both conditions. Until recently, the mental states involved in hypnosis have been little understood; the subjects have been supposed to be automata, wanting in volition and consciousness of self. The contrary, however, is the case; volition and memory are increased in hypnosis, consciousness of self is as vivid as in the normal state, and the general intelligence is often improved. It is these phenomena which demand an explanation, not the suppositious ones, which are based on mal-observations. Starting from erroneous premises, many authorities have attempted to explain the phenomena of hypnosis by an inhibition of some form or another of mental activity, and Hyslop's theory itself is simply a very old and well-known one masquerading in a novel garb. Indeed, it was clearly stated by John Hughes Bennett in 1851.

Bennett's Physiological Theory.—According to Bennett, hypnosis was characterised by alterations in the functional activity of the nerve-tubes of the white matter of the cerebral lobes. He suggested that a certain proportion of these became paralysed through continuous monotonous stimulation, while the action of others was consequently exalted. As these tubes connect the cerebral ganglion cells, suspension of their functions was assumed to bring with it interruption of the connection between the ganglion cells.

Bennett's Psychical Theory.—From the psychical side he explained the phenomena of hypnosis by the action of predominant and unchecked ideas. These were able to obtain prominence from the fact that other ideas, which under ordinary circumstances would have controlled their development, did not arise, because the portion of the brain with which they were associated had its action temporarily suspended, *i.e.*, the connection between the ganglion cells was broken, owing to the interrupted connection between the fibres of association. Thus, he says, the remembrance of a sensation can always be called up by the brain; but under ordinary circumstances, from the exercise of judgment, comparison and other mental faculties, we know it is only a remembrance. When these faculties are exhausted, the suggested idea predominates, and the individual believes in its reality. In this manner, we attribute to the faculties of the mind, as a whole, a certain power of correcting the fallacies, which each one of them is liable to fall into, in the same way that the illusions of one sense are capable of being detected by the healthy use of the other senses. There are illusions mental and sensorial; the former caused by predominant ideas, and corrected by proper reasoning; the latter caused by perversion of one sense, and corrected by the right application of the others.

In hypnosis, then, according to this theory, a suggested idea obtained prominence and created mental and sensorial illusions, because the check action—the inhibitory power—of certain higher centres had been temporarily suspended.

Bennett's theory, before it was finally enshrined by Hyslop in charmingly simple scientific phrase, had prac-

tically served the turn of all those who regarded the hypnotic personality as a limited or degraded form of the normal one. Thus, according to Heidenhain, the phenomena of hypnotism owed their origin to the arrested activity of the ganglion cells of the cerebral cortex. Because these were inhibited, sensory impressions, which usually produced movements, after passing to the higher centres and evoking consciousness, now did so by passing directly to the motor centres. This theory was accepted by Professor McKendrick, of Glasgow, and restated by him in the last edition of the "Encyclopædia Britannica," as giving a true and scientific explanation of the phenomena of so-called animal magnetism. Many objections have been successfully raised to this theory, while the supposed facts upon which it is based have been amply demonstrated to be without foundation.

Heidenhain simply reproduces the physical side of Bennett's theory, while Bernheim, although he denies all physiological change in hypnosis, faithfully reproduces the corresponding psychical one.

Bennett's theory crops up again in Vincent's "Hypnotism." Thirty-two pages are devoted to "neurons" and "dendrons," "inaptic" and "aptic" acts and reasons for rejecting the term "reflex." Finally we arrive at the hypnotic theory, which is founded simply on an inhibition of one set of functions—with an increased capacity of action in the others—the inhibition and dynamogeny of Brown-Séquard. "The stimulus," Vincent says, "instead of being dissipated amongst an indefinite number of neuronic groups, is confined to those whose function is the pure appreciation of the stimulus; the other groups, whose function is the consideration of the reasonableness of the suggestion, and the development of the stimulus in other conscious directions, are inhibited."

Sidis, in the "Psychology of Suggestion," explains the phenomena of hypnosis in exactly the same way. "There is," he says, "a functional dissociation between the nerve cells. The association fibres that connect groups into systems, communities, clusters, constellations—contract. The fine processes of the nerve cells, the dendrons, or the

termination, arborisation, or the collaterals that touch these dendrons, thus forming the elementary group—retract and cease to come into contact." He further discusses which association fibres give way first, and whether the neuraxon is contracted as a whole or whether the fibrillæ alone contract and so withdraw the terminal arborisations for minute distances. All this might be of interest if it were related in any way to the subject in dispute. The phenomena to be explained, however—increased memory, intelligence, &c.—are just the exact opposite of those which have been assumed, and theories, no matter how elaborate, nor how learned in terminology, are valueless when founded upon imaginary mental states, the existence of which is simply assumed by the operator. What does it matter whether lack of consciousness or loss of memory be produced by interruption of association fibres, arrested action of ganglionic cells of the cerebral cortex, retracted dendrons or disconnected neurons, or even by *an inhibition of the amœboid movements in the pseudopodic protoplasmic prolongations of the neurospongium*, if the problems we are dealing with are actually an increase of intelligence, consciousness, volition and memory?

Janet's Theory.—According to Pierre Janet, the secondary self is always a symptom of hysteria, while the essential fact about hysteria is its lack of synthetising power, and the consequent disintegration of the field of consciousness into mutually exclusive parts. Further, the secondary and primary consciousness added together never exceed the normally total consciousnesses of the individual.

Here the generalisation is certainly far too wide. Doubtless many cases of secondary consciousness correspond with Janet's description, others, however, present conditions which are its exact opposite. Thus, while hysteria is a prominent symptom in cases of spontaneous secondary consciousness, it is, on the other hand, not necessarily connected in any way with hypnotic form. Thousands of healthy men, absolutely devoid of any trace of hysteria, have been hypnotised, while hysteria itself, instead of favouring the induction of hypnosis, renders it more difficult.

Further, as to the relation between the severed personalities and the normal total consciousnesses, James justly points out that many instances have been noted, in which the secondary self is more highly developed than the primary one. It knows, for example, all that the ordinary self ever knew, and also much that the other had forgotten, or never known. Dr. Prince's case is a typical example of this. Here, the third personality not only knew all about her own life and that of the secondary personality, but also could recall (*a*) all that the normal personality could remember, and further (*b*) many things that the primary personality had known but forgotten, and (*c*) acts which the normal self had performed automatically, or absent-mindedly, and which had only reached normal consciousness partially, if at all; (*d*) finally, the third personality recalled events which had occurred in the delirium of fever, things of which the primary self was absolutely ignorant, and indeed had never known.

None of the theories we have been examining can be considered satisfactory. No revival of powers supposed to be possessed by lower animal forms can possibly explain the entire range of hypnotic phenomena; neither, again, can they be accounted for by an interrupted connection between the nerve cells, nor by that elastic term hysteria. Gurney's theory that the secondary consciousness calculates and watches the time, just as the primary one might do, is satisfactory for those instances in which it has been proved that this has occurred. It fails, however, when we attempt to apply it to those in which the facts were absent, and in which the feats in calculation and time appreciation were beyond the powers of the secondary self.

As the secondary self is often intellectually superior to the primary one, though we don't know why, we might be inclined to admit, although equally without reason for it, that the third self transcends the second one. As all the selves inhabit one body, and as the secondary and tertiary ones have presumably access to all the information which reaches the primary, are we to suppose that they have proved the apter scholars? It seems more than fanciful

to imagine that while the schoolboy's ordinary self was learning his lessons, a second boy was peering over his shoulder, as it were, and doing the work still better. What must we say, however, to the idea of a third boy learning along with Nos. 1 and 2, and surpassing both of them?

The examples, of time appreciation, we have been considering, present problems still more difficult. As we have seen, there is a marked difference between knowing a particular day on its arrival, and recognising which is the terminal one amongst a long series of minutes. For example, suppose that a suggestion to be fulfilled in 20,840 minutes fell due at 3.15 on a certain afternoon, and that from 12.20 p.m. of the same day the subject of it remained in a room, where she was deprived of all ordinary means of determining the passage of time. Granting that the subject's third personality had calculated 3.15 as the terminal time, that she had noted the time on entering the room and calculated that 3 hours and 55 minutes had to expire before the suggestion was due, and even admitting that she was able to impart this information to her primary consciousness, and to enlist its aid in her attempt to hit upon the exact moment, viz., 3.15, the question still remains, how was this done?

According to William James, our usual time appreciations—minutes, hours and days—have to be conceived symbolically and constructed by successive mental additions. To realise an hour, we must count "now!" "now!" "now!" indefinitely. Each "now" is the feeling of a separate bit of time, and the exact sum of the bits never makes a very clear impression on our minds. We have no sense for empty time and have to subdivide the time by noticing the sensations; after we have received a certain number, our impression of the amount told off becomes quite vague, and our only way of knowing it accurately is by counting, noticing the clock, or some other symbolical conception. Our estimation of the length of time varies from many causes. A time full of interesting experiences seems short in passing, but long when looked back upon; time empty of incident is long in passing, but seems short in

retrospect. Time passes quickly when we are so occupied with what is happening as not to notice the time itself. When we do nothing and feel little we grow more and more attentive to the passage of time. The length of a watched minute seems incredible, because the attention is devoted to the feeling of the time itself, such attention being capable of extremely fine subdivision.

In conclusion James says, "We are constantly conscious of a certain duration—the spacious present—varying in length from a few seconds to probably not more than a minute, and that this duration (with its content perceived as having one part earlier and the other later) is the original intuition of time. Longer times are conceived by adding, shorter ones by dividing, portions of the vaguely bounded unit, and are habitually thought by us symbolically. Kant's notion of *intuition* of objective time as an infinite necessary continuum has nothing to support it. The *cause* of the intuition, which we really have, cannot be the *duration* of our brain processes or our mental changes. That duration is rather the *object* of the intuition which, being realised at every moment of such duration, must be due to a permanently present cause. This cause—probably the simultaneous presence of brain processes of different phase—fluctuates; and, hence, a certain range of variation in the amount of the intuition and its subdivisibility accrues."

Further, "the direct *intuition* of time is limited to intervals of considerably less than a minute. Beyond its borders extends the immense region of *conceived* time, past and future, into one direction or another of which we mentally project all the events which we think of as real, and form a systematic order of them by giving to each a date."

If we accept James' explanation as to the methods by which time appreciations are carried out—and I know of no other more plausible—the time appreciations of Miss A. seem still more remarkable and inexplicable.

While I have endeavoured to show that the theories as to the origin of secondary and multiple personalities—both as to the states themselves and their phenomena—are entirely inadequate, I have no theory of my own to bring

forward in substitution for these. The fresh facts I have cited, although interesting, only add to the complexity of the problems we have to solve. As William James truly says, these manifestations of the "hidden self" are immensely complex and fluctuating things, which, we have hardly begun to understand and concerning which sweeping generalisation is sure to be premature. Meanwhile, he adds, a comparative study of subconscious states is of the most urgent importance for the comprehension of our nature.

HEMIATROPHY OF THE BRAIN AND ITS RESULTS ON THE CEREBELLUM, MEDULLA, AND SPINAL CORD.

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THIS paper deals with three cases of partial atrophy of one cerebral hemisphere. The lesions are different in each case, both in regard to their situation and nature; but they readily group themselves into two classes, and this may be said of the numerous other cases of porencephaly, &c., which have been previously described. These groups are :—

(1) *Cortical.*—In which the lesion is confined to the cortex or underlying white matter. (Two of the cases to be described are of this nature.)

(2) *Basal.*—In which the original lesion is situated in the structures at the base of the brain, as occurred in the remaining case.

With these two varieties are associated secondary changes in other portions of the cerebro-spinal axis, and we believe that different changes occur when the lesion is confined to the cortex, and when the basal ganglia are also affected. It is proposed to first give an account of the examination of each case, and then to endeavour to point out these differences.

Case 1.

Primary lesion of left basal ganglia (probably optic thalamus) occurring at a very early age, and probably due to occlusion of a basal perforating artery. Atrophy of left cerebral hemisphere, including basal ganglia, of left crus, left pyramid and fillet in pons and medulla. Atrophy of right half of cerebellum with its superior peduncle. Sclerosis of left direct and right crossed pyramidal tracts in cord. Atrophy of left antero-lateral column. Diminution in number of anterior horn cells in cervical and lumbar regions, affecting the lateral and postero-external groups. Clinically—epileptiform fits from infancy. Right hemiplegia with increased reflexes. Progressive dementia. Death at the age of 30 *years.*

Clinical History.

J. E., male, died in Claybury Asylum at 30 years of age of acute phthisis. Since infancy he had been subject to fits, and mentally was rather dull and stupid. At the age of 26 he became subject to attacks of maniacal excitement, so that he had to be sent to the asylum. He was then found to have right hemiplegia with increased reflexes (considered to have dated from infancy). He had frequent fits of an epileptiform type whilst under observation, and gradually became more and more demented until his death.

Post-mortem Examination.

The bones of the right hand, arm and leg are obviously smaller than the left, and the muscles are much atrophied.

The *Skull* shows no change externally, but on examining it from the inside the whole of the left half is found to be very much thicker; this is most marked in the frontal and parietal regions. Thus the thickness of the bone over the left frontal lobe is 20mm. as compared with 11mm. of the opposite side. Over the left parietal and central regions it is 12mm. as compared with but 5mm. of the opposite side. The base of the skull in the anterior and middle fossæ is also thicker on the left side. In the occipital region there is comparatively little difference between the two sides, and this is in correspondence with the fact that in this region there is but little atrophy of the brain substance. This overgrowth of bone is undoubtedly secondary to, and corresponds with, the atrophy undergone by the brain.

The *Membranes*.—Beyond some slight thickening of the piâ over the wasted hemisphere, these show no change of note. There is a considerable quantity of cerebro-spinal fluid present at

the base of the brain, but no abnormality of any of the vessels could be discovered.

Brain.—There is a notable difference between the size of the two hemispheres. The weight of the left is but 155 grams, whilst that of the right is 575 grams. This latter weight may be taken as normal, and as no microscopical change could be discovered on examining the right hemisphere, it may be dismissed from further consideration. On a further examination of the left hemisphere, it is found that the diminution in weight is due to a marked atrophy of the white and gray matters, the ventricle being hugely dilated in all its parts. The basal ganglia, and especially the optic thalamus, are also quite insignificant as compared with the opposite side. It is noticed that as well as the convolutions being so extremely wasted, they are simpler in nature than on the opposite side. All parts of the hemisphere are not equally affected, however; the greatest amount of atrophy exists in the central and parietal regions, next in the frontal, and least of all in the occipital. This fact is shown not only by the appearance, but also by a comparison of the weights of the different regions. There is no difference in size between the optic tracts nor the olfactory lobes of the two sides. The corpus callosum is much thinner than normal.

Portions of the cortex were removed from the frontal, central, and occipital regions, and after staining by different methods were examined microscopically with the following results:—

Frontal and central regions.—It is here that the greatest change is found. The outermost layer of cells and the layer of small pyramids are fairly normal; the large pyramids, however, are very deficient, indeed, they are practically absent, and the cells below these are also much diminished in number. With regard to fibres, there is an almost complete absence of those of the projection system, and there are also very few of either the superficial tangential fibres or of those composing the line of Baillarger. The medullary centre consists chiefly of bundles of the longer association fibres, and generally it is found that this system is least of all affected, the tangential system to a slightly greater extent, and the projection fibres most of all. The great diminution of the large pyramids and their axons is evidently a secondary change resulting from the basal lesion, the fibres being probably originally affected in the internal capsule. It is thus of the nature of a Gudden atrophy. As will be presently seen in the examination of the cord, the distal portion of the efferent tract is also greatly affected (fig. 2).

Occipital region.—The cortex of this region has undergone very little change, the difference between this and other parts being very striking. All the layers of cells can be made out, and the tangential fibres and line of Gennari are well seen. It may be here stated that the patient had no affection of sight, and that the posterior portion of the thalamus and the optic commissure and tracts appeared normal (fig. 1).

The *Basal ganglia* are much smaller than on the opposite side; the greatest difference is in the anterior two-thirds of the thalamus, where the number of cells is much diminished. The internal capsule also contains very few medullated fibres.

Pons.—There is marked atrophy of the pyramidal fibres and also of the middle fillet on the left side.

The *Medulla* is also markedly asymmetrical, chiefly due to the small size of the pyramid and interolivary fillet of the left as compared with the opposite side. In the lower portion of the medulla there is also a slight sclerosis dorso-lateral to the left olive (in the region occupied by the antero-lateral column). There seems also to be some slight diminution of the left external and right internal arcuate fibres.

No differences can be made out between the posterior column nuclei of the two sides, nor between the restiform bodies, the olives, nor the posterior longitudinal bundles, all these appearing to be quite normal.

Owing to the method which had been used for hardening the tissue, an examination of Deiter's nucleus by means of Nissl's method could not be made; but we could not discover any appreciable change between the two sides in carmine-stained sections. The changes in the pyramid and fillet do not appear to be of the kind one finds in secondary degeneration with resulting sclerosis; they consist of an actual diminution in the number of fibres, with slight interstitial overgrowth, the fillet being but half the size of the opposite one. The microscopical appearances are more like those found in the arrested development of a tract than due to degeneration occurring in a tract already fully formed. It has been seen that the lesion probably occurred before the myelination of the efferent system, and it is easy to see how a lesion at the base of the brain would involve these motor fibres.

The reason why the fillet should be so much atrophied is not at first sight so apparent. It may be thought to be due to an atrophy of fibres having an ascending direction; there is, however, no change in the posterior column nuclei. It has been seen that in the lower part of the medulla there is slight sclerosis in that

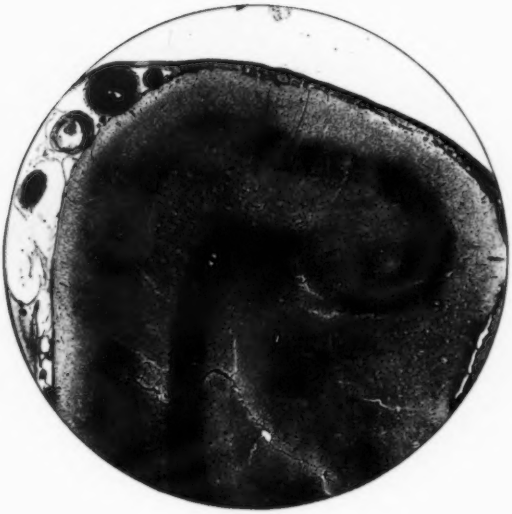

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

portion occupied by Gowers' tract, and it will be shown presently that there is very extensive atrophy in the antero-lateral column of the cord on the same side. Although some fibres from Gowers' tract undoubtedly ascend through the region of the fillet, these fibres are comparatively few in number, and would not account for a reduction of the fillet to half its normal size. Indeed, as will be presently seen, it is likely that the atrophy of the antero-lateral column of the cord is not due to Gowers' tract at all.

The more probable explanation of the wasting of the fillet is that it is the result of atrophy of *descending* fibres, probably having their origin in the thalamic region. There is strong evidence that there are descending fibres passing through the middle fillet of the pons and the interolivary fillet of the medulla. It will, perhaps, be better to return to this subject after the condition of the cord has been described.

Cerebellum.—Weight of left half 70 grams, of right half 55 grams. Atrophy is found in the right half only (the side opposite the cerebral lesion), and on this side it is confined to the following situations:—(1) The lobus culminis, which is affected throughout its entire extent; (2) the biventral lobule, the posterior half only of which is atrophied. The dentate nucleus is also much smaller, and contains fewer cells, although what are present appear to be normal. The superior peduncle on this side is also only a quarter the thickness of the opposite one. There is no change in the vermis nor any other part of the cerebellum.

With regard to the particular structures affected by this atrophy, there are a few points of great interest:—(1) In the atrophied region there is an entire absence of the cells of Purkinje; (2) it can be readily made out (owing to the selective nature of the changes) that the granule layer is divisible into two strata of cells.

Immediately beneath the layer of Purkinje's cells there is a layer of round cells, two or three deep, which are distinctly larger than those making up the bulk of the granules. This outer layer persists unchanged in the atrophic lobules. Normally, the deeper granules, which may be called the granules proper, are much more numerous and are smaller than these cells; indeed, they are so numerous that it is difficult to distinguish the outer layer in the normal cerebellum as it is overlaid by the granules. In this case, however, the true granules are profoundly diminished, in some places almost absent, and thus the more superficial layer (remaining unaffected) comes readily into view. The

molecular zone is also thinner in the affected region, and contains fewer cells. It would further appear that whilst the atrophy especially affects these two cell groups (Purkinje's and the true granules) Purkinje's are the first to suffer, as several of the leaflets show an intermediate stage, in which but two or three of these cells are present, whilst the number of granules is still considerable (figs. 3, 4, 5).

With regard to the fibres it is at once seen that there is a great diminution. This diminution is almost entirely confined to the *large* fibres of the medullary rays, which are presumably connected with Purkinje's cells. In the absence of these fibres it is seen that the medullary substance and the region of the granules are occupied by a network of fine interlacing fibres, which would seem to belong to an association system. Further, bundles of fibres can seen passing in the medullary centre between adjacent folia, and fibres having a tangential course can be seen in the deeper parts of the molecular layer, and in the region occupied by these large granules. In view of these facts the inference is that this layer of large granules, which are the only cells remaining unchanged, are those which are chiefly in relation with an inter-cerebellar association system (figs. 6, 7).

Golgi drew attention to cells of a large kind lying in the zone of granules, and he described them as having protoplasmic processes ending in this and in the molecular zone, and with an axis-cylinder process penetrating into the granule layer and there forming an inextricable network. Cajal (quoted by van Gehuchten) and van Gehuchten himself (6), also consider these cells and their processes as an association system.

The cerebellar atrophy in this case is owing to the connection of the cerebellum with the opposite basal ganglia by means of fibres passing through the superior peduncle. This connection is now well established by numerous observations, and probably takes place by the conjunction of several neuronic systems. There are two views as to the direction of these fibres; some authorities agreeing with Mahaim (12) that they have their origin in the red nucleus, and are thus cerebello-petal; whilst Cajal (4) and others think that they arise from cells situated in the cerebellum, and are thus cerebello-fugal. The bulk of the evidence seems to show that whilst fibres may pass through the superior peduncle in both directions, the majority have their origin in cells situated in the cerebellum, as after cerebral lesions the change observed in the superior peduncle is rather one of atrophy than of secondary degeneration. It is unfortunate that

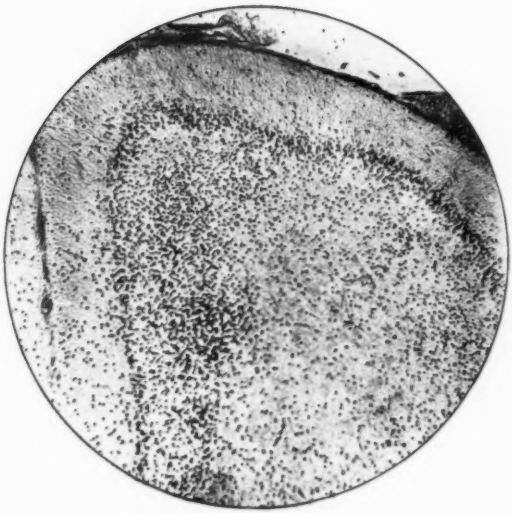

FIG. 5.

FIG. 6.

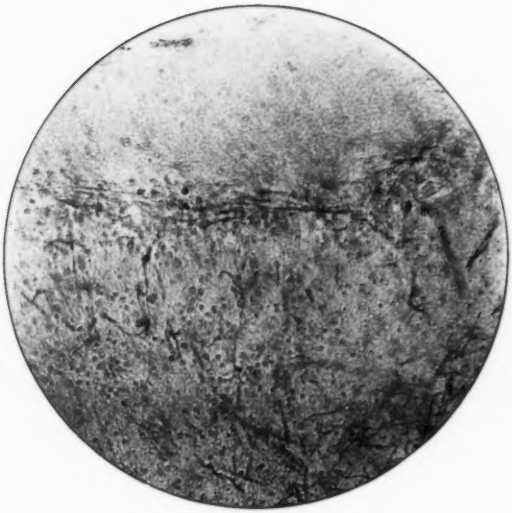

FIG. 7.

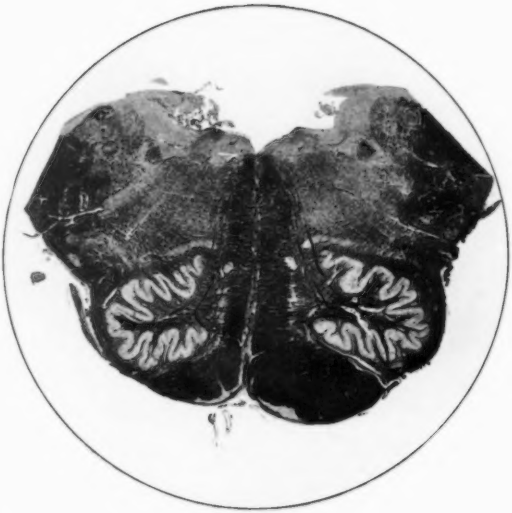

FIG. 8.

in this case the brain had been so cut that the red nucleus could not be examined. Opinions are also very much divided as to the actual origin of such cells within the cerebellum. Held (8) thinks that the larger number of such arise from the dentate nucleus. Menzel and Dejerine agree with him, finding that a lesion which involves the cortex and adjacent white matter only, does not lead to any marked change in the superior peduncle or red nucleus. On the other hand Marchi (18), Mingazzini (13) and Cajal (4) state that there *is* considerable degeneration in the fibres of the superior peduncle after extirpation of the cerebellar cortex; indeed, Cajal has shown (in the mouse) that the axones of Purkinje's cells go directly into the superior peduncle, and von Monakow (15) has recorded a case of atrophy of the right cerebrum and left cerebellum and its superior peduncle, with no change in the dentate nucleus.

The very considerable atrophy of the dentate nucleus in this case shows that fibres certainly pass between it and the opposite basal ganglia. The atrophy of the cerebellar cortex may be secondary to that of the dentate nucleus, or may be due to the involvement of other fibres passing directly to the basal ganglia without the intervention of the dentate nucleus.

Spinal cord.—This is obviously asymmetrical, the left half of the cord being much smaller than the right. The difference between the two sides gradually diminishes from above downwards. It is exceedingly well marked in all parts of the cervical region, and slightly less so in the dorsal; it can, however, be definitely made out even as far as the fourth lumbar segment. Below that level there is practically no difference between the two halves. There are two factors which bring about this asymmetry: (1) In the left and smaller half of the cord there is a well-marked atrophy at the periphery of the cord in the situation of the antero-lateral tract. The area of the crossed pyradimal tract on this side also appears to be smaller than usual, and certainly smaller than the corresponding area of the opposite side. (2) The anterior column on the right side is much larger than normal. In the photographs this is very apparent, it being readily seen that the anterior column to the right of the median fissure is almost double the size of that to the left in the cervical and dorsal regions. This inequality appears to be chiefly due, however, to an excessive size of the right, the left being only slightly less than normal. This fact, taken in conjunction with the smaller size of the left crossed pyramid, indicates that there has been an unequal crossing of the pyramidal tracts. That is to

say, that as a result of the lesion at the base of the brain interfering with the efferent fibres of one side, there is interstitial sclerosis of the right crossed and left direct pyramidal tract. And that of the healthy efferent tract coming from the *right* cerebral hemisphere a great many fibres do not undergo decussation in the medulla, but are continued down the cord on the same side, partly in the lateral column, but mainly in the anterior column, where they form an unusually large direct tract. There is also slight sclerosis of a portion of the crossed pyramidal tract on the same side as the cerebral lesion. This is well seen in the photographs and is undoubtedly due to the involvement of homolateral fibres.

It has been said that there is considerable atrophy of the left antero-lateral column. This is most marked at the periphery, and causes a very evident indentation of the cord as low down as the first lumbar segment. It is in the situation of the descending cerebellar path, but we do not think it is due to involvement of that path, because the homolateral cerebellar hemisphere and the vermis are perfectly normal, nor could any change be made out in Deiter's nuclei. The condition of the cerebellum also negatives the idea that the atrophy may represent the ascending tract of Gowers, as most of these fibres go to the vermis (17). The view we take, and which seems to best fit in with the case, is that this atrophy is in a *descending* path having its origin in the thalamic region. It is probable that in the medulla these fibres pass downwards through the fillet, and it has been seen there is marked diminution in the number of fibres in this region.

Hoche (10) has recently described two cases in which he found the fillet fibres degenerated in a descending direction, such fibres going to the cranial nerve nuclei, and he further states that such descending fibres may readily be distinguished in the new-born child on account of their late myelination. Flechsig and von Bechterew have also shown that a certain number of fibres in the region of the fillet become medullated at a much later date than do the bulk of the fillet fibres. In this connection it is of great interest to note that a descending (probably) tract has been seen passing between the lower part of the medulla and the upper dorsal cord by several observers. Its situation in the cord is in the most peripheral part of the antero-lateral column. Meyer (16) described degeneration in such a bundle in a case of pontine hæmorrhage. Helweg (9) described it under the name of "Dreikantenbahn," and von Bechterew has also described a tract which seems to be identical in position. Von Bechterew (2) calls it the "Olivenstrang." He seems to think that it is an ascend-

FIG. 9.

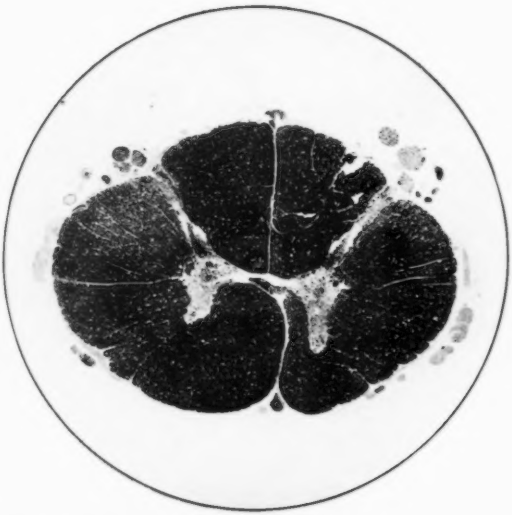

FIG. 10.

FIG. 11.

FIG. 12.

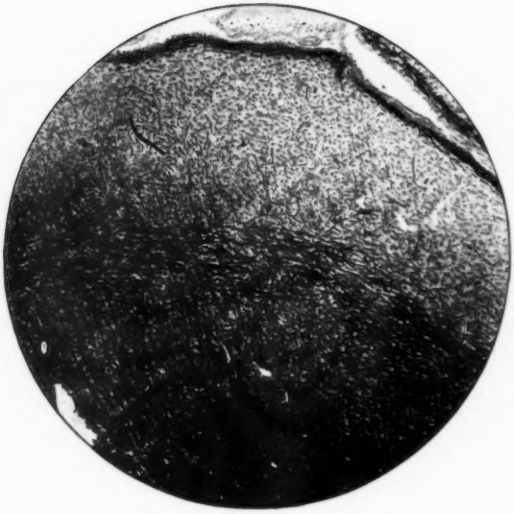

FIG. 13.

FIG. 14.

ing tract which enters into the olivary nucleus, as he has not been able to trace it above that point; he admits though that there is no proof that its fibres end there. Barker (1), who gives a very good account of this tract, has himself observed it. He is inclined to look upon it as a descending tract, and to consider that the fact that it may be degenerated whilst the olive is quite normal is against the view that it arises in that nucleus. He suggests that the fibres may come from higher parts of the nervous system, possibly being so scattered above the level of the olive that they are not readily distinguished.

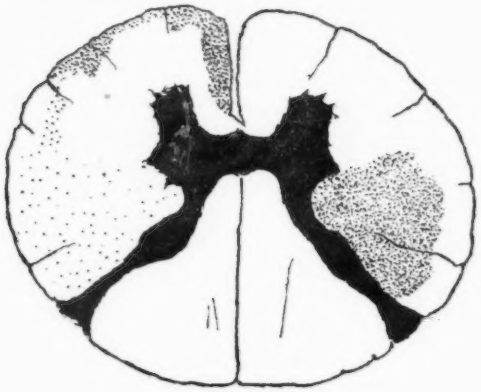

FIG. 15.

Spinal Cord.—Section at level of first cervical segment from a case of thrombosis of the left middle cerebral artery. Marchi stain. In addition to the degeneration in the direct and crossed pyramidal tracts, there is also degeneration in the antero-lateral column at the periphery of the cord on the *same* side as the cerebral lesion, also scattered degeneration (homolateral fibres) in the lateral pyramidal tract of the same side.

In this place we wish to refer to another case which we have recently examined.

Patient was affected rather suddenly with right hemiplegia. He was admitted into hospital, but died within three weeks of the onset of the first symptoms.

At the autopsy the left middle cerebral artery was found thrombosed at its commencement. No change was discovered in any of the other vessels. The greater part of the spinal cord was used for a chemical estimation, but the upper cervical region was

put into Marchi's fluid and examined microscopically. In addition to the ordinary degeneration in the corresponding direct and crossed pyramidal tracts, these sections show well-marked recent degeneration of a bundle of fibres at the periphery of the cord in the antero-lateral column of the same side as the cerebral lesion; there is also degeneration of a few fibres passing dorsalwards from this towards the lateral pyramidal tract, but in this case the degenerated fibres are a little distance from the periphery of the cord. There is also scattered degeneration throughout the lateral column of the same side, doubtless due to homolateral fibres. The extent and position of the degenerated fibres are well shown in fig. 15, which is a drawing made by the Edinger apparatus, the finer details being filled in under the microscope.

In this case the degeneration in the antero-lateral column is identical in position with the change in Case 1. No other lesion than that of the middle cerebral artery could be discovered, it therefore seems fair to consider that the degenerated fibres in the antero-lateral column have their origin from cells within the area of distribution of this vessel. So that hitherto, although several observers have noticed the existence of such a tract in the antero-lateral column of the cord in the cervical region, its upward continuation above the olive and downward continuation below the cervical portion of the cord have not been shown.

We are inclined to think that the atrophy of the antero-lateral column in this case is due to the involvement of this tract; that its probable origin is in the optic thalamus; that it passes through the middle fillet in the pons and the interolivary lemniscus in the medulla to reach the peripheral part of the antero-lateral column of the cord, in which it descends as far as the lumbar region.

It has been conjectured that a descending thalamo-spinal path exists in man, although no direct proof has so far been afforded. It is well known, however, that in several of the lower animals with but little cortico-spinal connection there is a very well developed thalamo-spinal tract. We know that in man a very large number of the sensory fibres end in this region. Most of the ascending fibres of the fillet (coming by means of the posterior column nuclei from the columns of Goll and Burdach) end in the thalamus; the fillet also receives fibres from the antero-lateral ascending tract. Thus the thalamus is a large receiving station on the afferent tract, and by means of these efferent fibres just described, it becomes a large and important ganglionic station midway between the lower spinal reflexes and the higher cortical volitional centres, and probably functionating in those multi-

tudinous semi-automatic movements which do not rise into consciousness.

Further support of this view of the importance of the thalamic region as a ganglionic station is afforded by the well known case of Goltz's dog. In this animal both cerebral hemispheres were removed, but the co-ordination of the most complicated automatic movements remained.

It may here be mentioned that we have examined the cells of the optic thalamus of a new born child by means of Nissl's method, and we find that whilst there are several distinct varieties a large number of them are evidently motor. Such cells are intermediate in size, shape, and period of development, between the motor cells of the anterior horns and the large pyramidal cells of the motor cortex.

So far no efferent tract passing directly between the cerebellum and the spinal cord has been satisfactorily demonstrated, and the researches of Ferrier and Turner (5) and Risien Russell (21) do not support Marchi's views as to the existence of such direct connection. There is certainly no evidence in this case to support the view of any such connection, unless the atrophy of the *opposite* antero-lateral column of the cord can be considered to be due to cerebellar fibres.

In view of these findings we suggest the possibility of cerebellar fibres to the cord passing by way of the basal ganglia (possibly the optic thalamus).

Ganglion cells.—In the cervical and upper dorsal regions it is evident to the naked eye that the right anterior horn has a shrunken appearance and is smaller than the left. On examining Nissl-stained sections, important changes in the ganglion cells are found. Above the fifth cervical segment there is practically no difference between the two sides; but at this level there are undoubtedly fewer cells present on the right side, and the diminution is almost entirely confined to the cells in the lateral and postero-external groups. At the eighth cervical and first dorsal segments there are identical changes affecting the same cell groups. The mesial anterior group of cells appears to be quite normal and equal on the two sides. In the upper part of the second dorsal segment similar changes are found in the right lateral group; but at this level there is unquestionably a diminution in the cells of the mesial-anterior group of the *left* side. A series of Nissl-stained sections were examined to ascertain the condition of the cells, it being quite impossible to make any reliable observations upon diminution of cells from the examina-

tion of only a few sections. To give an idea of the extent of the change we may say that in sections through the eighth cervical and first dorsal segments the number of cells in the right lateral and posterior groups was but half those in the same groups of the opposite side. In the dorsal region sections were examined from the fifth, ninth, tenth and twelfth segments. At none of these levels are there any differences between the anterior horn cells of the two sides.

The limitation of the changes to these segments is in accord with the observations of Ferrier and Yeo, Risien Russell, Allen Starr and others, who find that the muscles of the arm, forearm, and hand are enervated from the fifth cervical to the first dorsal segments inclusive. This case also shows that it is chiefly the lateral and postero-external groups of cells in these regions from which the nerves are derived, and is in accord on this point with our findings in cases of amyotrophic lateral sclerosis in which the same cell groups are those chiefly affected. In this respect the view of von Lenhossék (11) is worthy of note. He considers that the mesial-anterior group of cells are not motor but commissural in function, sending their axones through the anterior commissure to the opposite side of the cord. It is believed by Kaiser and others, however, that this group of cells supplies the muscles of the back, but from this case they certainly appear to have but slight connection with the cortex.

In the tenth and twelfth dorsal segments there is a slight diminution in the number of cells composing the right Clarke's column, and a similar change is also seen at the level of the first lumbar segment. In the lumbar and sacral regions there is again marked diminution in the motor cells of the right anterior horns, and here, too, the changes are chiefly confined to the lateral and posterior groups of cells, the mesial-anterior group showing no change. The changes here described are also seen very well in carmine-stained sections; they consist in an actual diminution of ganglion cells in certain groups, and although in the affected group some of the remaining cells are atrophied, the change is principally a quantitative and not a qualitative one.

Von Monakow (15) believes that the fibres of the pyramidal tract are not in immediate contact with the lower motor neurones, but that there is another neuronic system interposed. Schäfer (22) holds the same opinion. He finds that after lesion of this tract no degenerated fibres can be traced directly into the anterior horns, or into any part of the gray matter other than the base of the posterior horn and Clarke's column.

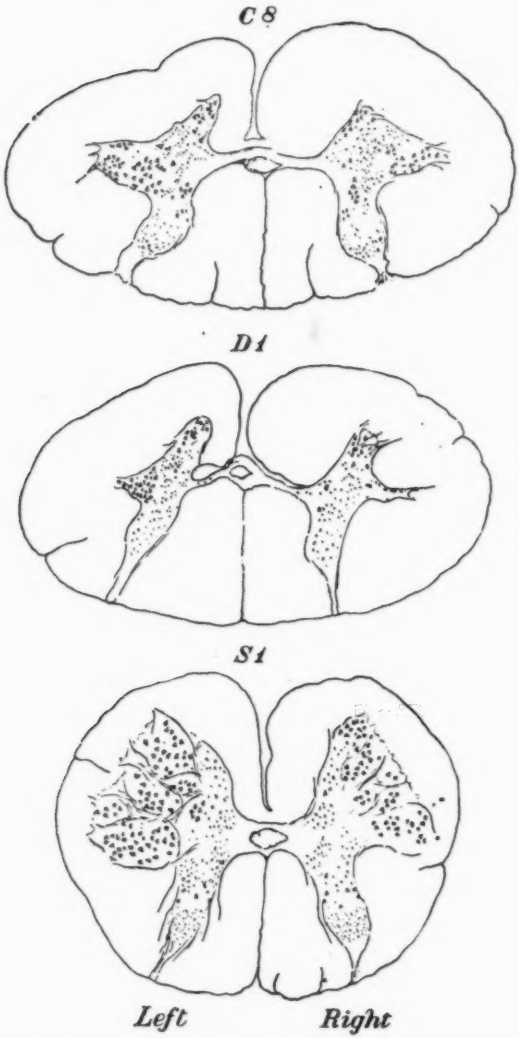

FIG. 16.

Spinal Cord.—Three drawings, by means of the Edinger apparatus, of Nissl-stained sections from case 1, showing the diminution in the number of cells in the cervical and lumbar regions, almost entirely confined to the lateral and postero-external groups.

It may be that another neurone is interposed between the pyramidal tract and the anterior horn cells; but it does not appear from this case that such junction is by means of Clarke's column, and Rothman (20) denies that the fibres of the pyramidal tract terminate in that column.

In sections through the cervical and lumbar enlargements stained by the Weigert-Pal method, there is seen to be not only a diminution in cells in the anterior horns, but also a far less plentiful network of fibres than in the normal cord, especially is this so in the centre of the horn. This rather suggests that the fibres of the pyramidal tract may here break up into a fine arborisation, and so come into immediate connection with the motor cells.

Case 2.

Atrophy of left hemisphere, involving the cortex only around the central convolutions. Basal ganglia not affected. Atrophy of corresponding efferent tract in pons, medulla and cord. No change in fillet or cerebellum. Well marked tangential, super, and inter-radial fibres. Numerical diminution of anterior horn cells in cervical and lumbar regions. Clinically—right hemiplegia from birth with (?) epileptic fits. Exaggerated knee-jerks. No dementia. Death at the age of 33 *years.*

Clinical History.

J. H. has had right hemiplegia from a very early age (probably from birth). He has also been subject to fits (? epileptic) since he was a fortnight old, but he was apparently able to partially earn his living until 28 years of age. At this age, owing to the severity and frequency of his fits, and his impulsive, violent, and unreliable mental condition, he had to be sent to an asylum. Whilst here he had frequent fits, but was able to do a fair amount of useful work in between them. At the time of his death (aged 33 years) he showed comparatively little mental degradation. We saw him a short time before his death, and his condition was then as follows:—There was paresis of the right hand and arm, the finer movements of the fingers being especially affected (opposition, abduction and adduction). This hand was blue and cold, and the muscles of the thenar and hypothenar eminences and the interossei were decidedly less developed than on the opposite side. Occasional athetoid movements were

observed to take place in this hand. There was also paresis of the right foot, the finer movements here being most affected, and the muscles of the right calf were much less developed than those of the left. There was no paralysis of any muscles supplied by cranial nerves. The knee-jerks were exaggerated but equal on the two sides. No ankle clonus. Plantar, cremaster and abdominal reflexes were normal. The pupils were equal and reacted to light and accommodation. Cutaneous sensation appeared to be normal all over the body.

Post-mortem Examination.

The right hand and arm and the right leg are shorter and smaller than those of the left side.

The *Skull* is asymmetrical as seen from the outside, the left parietal eminence being decidedly less prominent than the right, and the skull being somewhat flattened in this region. There is, however, no difference in the thickness of the bone on the two sides, neither of the vertex nor the base.

Membranes.—The dura mater is normal. The piâ-arachnoid is opaque and thickened and of greatly increased vascularity over a considerable area on the external surface of the left hemisphere. This area comprises the following convolutions: The whole of the ascending frontal and ascending parietal, and the posterior portion of the superior frontal, the convolutions above and in front of the inter-parietal sulcus as far back as the parieto-occipital fissure. It is thus somewhat wedge-shaped in appearance, with the base of the wedge at the great longitudinal fissure and the apex at the anterior part of the sylvian fissure. Over this area the piâ is closely adherent and cannot be removed without causing decorticication. In the other parts of the brain it is perfectly natural in appearance.

Brain.—The left hemisphere weighs 105 grams less than the right. It is shorter and smaller as a whole, but there are no cyst-like depressions and no dilatation of the ventricles. The main sulci are somewhat distorted in their arrangement and position, but with a little difficulty they can all be traced. The right hemisphere is perfectly normal in every way. The basal ganglia are unaffected and equal on the two sides. There is no change in the corpus callosum, in the olfactory bulbs, nor in the optic commissure or tracts. Portions of the cortex from several regions of the brain were examined microscopically. All those situated outside the area above described were quite normal. Within this area

sections have the following appearance:—At frequent intervals there are foci in which the nerve-cells are considerably diminished in number and much jumbled up in arrangement. In these situations there are no definite Meynert's columns or even readily distinguishable layers. There is also a considerable increase of neuroglia cells and of leucocytes, and the vessels are much distended. The portions intervening between these areas approach much more nearly to the normal, but even in these there is a deficiency of large nerve-cells. Throughout the whole of the motor region affected there are comparatively few of the larger pyramids and of Betz's cells. The fibres of the projection system are also much diminished, but the tangential and inter-radial fibres are present in abundance (figs. 13, 14). A section from an exactly corresponding region of the opposite healthy hemisphere shows large numbers of Betz's cells, in fact, they are present to almost greater extent than we have ever observed before, thereby suggesting that a compensatory increase has taken place.

In the *Crura* the left pes is very much smaller than the right; but the red nucleus is unaffected.

In the *Pons* the left half is much smaller than the right, and there is great diminution in the longitudinal efferent fibres. There is no change in the fillet.

The *Medulla* is also much smaller on the left side, owing to great diminution in the size of the pyramid. This contains fewer nerve fibres and there is some interstitial sclerosis. The olives, the interolivary fillet, and other portions of the medulla show no change.

The *Cerebellum* is quite healthy, and the two halves weigh exactly the same.

The *Spinal cord* is asymmetrical, owing to a diminution in the number of fibres of the left direct and the right crossed pyramidal tracts. In these tracts there is slight interstitial sclerosis, but the fibres in the sclerosed tracts do not appear to be smaller than those on the healthy side. The change in the direct tract ceases to be visible below the mid-dorsal region, whilst that in the crossed tract can be traced as far as the sacral region. There is no change in the antero-lateral columns, the posterior columns, nor any other portion of the white matter. Marchi stained sections do not reveal any recent degeneration. In sections through the cervical and lumbar enlargements there is an alteration in the motor cells of the anterior horns similar to that found in case No. 1, although in this case such changes

are not quite so pronounced. They consist in a diminution in the number of cells composing the lateral and postero-external groups on the right side, and in the same regions the network of fibres is also much less abundant. This diminution in the network is especially pronounced in the middle of the anterior horn, there seem to be as many fibres as usual towards the periphery of the horns. No change can be made out in the mesial-anterior group of cells. The cells of Clarke's column are plentiful, equal on the two sides, and appear perfectly normal.

Case 3.

Atrophy of convolutions of lower part of motor region on left side, and of corresponding efferent tract in pons, medulla and cord. No change in basal ganglia, fillet or cerebellum. Clinically —Right hemiplegia from birth. Epileptiform fits from 12 *years old. Progressive dementia. Death at the age of* 22 *years after severe fits.*

Clinical History.

A. W. was born paralysed on the right side. She had epileptiform fits from 12 years of age, and then began to complain of headache. At the age of 15 mental symptoms made their appearance, and when 18 years of age she had to be sent to the asylum, being then described as dull, listless, and apathetic, with a weak mind and memory, and hesitating speech. She was never able to follow any regular employment. When admitted she had paresis, with wasting of the right arm and leg. Pupils were equal and reacted to light. Knee-jerks were absent. Speech indistinct. No paralysis or affection of the facial muscles. Whilst in the asylum she was subject to constant fits, these began on the paretic side and then became general. She gradually became more and more demented, and died at the age of 22, after a succession of severe fits.

Post-mortem Examination.

The right arm and leg are shorter than the left, there being a difference of one and a half inches between the girth of the calves, two inches between the thighs, and one inch between the forearms of the two sides.

The *Skull* is asymmetrical, the left side being smaller and thinner than the right. There is an opaque thickening of

the piâ-arachnoid over the affected area of the brain described below.

Brain.—Weight of left hemisphere 435 grams; weight of right hemisphere 585 grams. There is a small depression in the brain substance at the lower portion of the left motor region, filled with clear fluid and having the thickened piâ stretched over it so as to form a kind of cyst. This depression is due to shrinkage of the lower two-thirds of the ascending frontal and ascending parietal, and the posterior portion of the third frontal convolutions. The left ventricle is greatly dilated. The right hemisphere and all other portions of the brain are normal. No difference exists between the basal ganglia on the two sides.

In the *Pons* there is marked atrophy of the longitudinal bundles of the left side causing considerable asymmetry. No change can be made out in the fillet.

The *Medulla* is also very asymmetrical, the pyramidal fibres on the left side being reduced to a very thin strand. No change can be found in any other region. The olives and the interolivary fillet appear quite normal and of equal dimensions on the two sides.

The *Cerebellum* shows no change whatever. The two halves weigh exactly the same.

The *Spinal cord.*—Sections of the cord were examined at the levels of the second, fifth and sixth cervical, tenth, eleventh and twelfth dorsal, and first lumbar segments. In all these sections it is seen that the right half of the cord is smaller than the left. On careful examination it is quite apparent that this is simply due to diminution of the right crossed pyramidal tract. Under the microscope this area is found to contain fewer fibres and to be the seat of slight interstitial overgrowth of neuroglia. In the cervical region this slight sclerosis extends in the form of a tail-like process almost as far forwards as the origin of the motor roots. In the dorsal and lumbar regions it is confined to the usual triangular-shaped area of the pyramidal tract. In the region of the direct tract there is slight sclerosis and diminution of nerve fibres on the left side as far down as the fifth cervical segment. There is no microscopical change, nor any want of symmetry between the antero-lateral columns, nor the posterior columns, nor any other portion of the white matter of the cord.

In this case, therefore, although the pathological process probably differs from case No. 2, the anatomical site of the lesion is very much the same, and the results on the medulla, cerebellum and spinal cord are practically identical in the two cases.

Remarks.

It is not proposed to discuss at any length the pathology of these cases. There are probably many causes which may bring about such conditions; but it is likely that vascular occlusion, arterial or venous, is responsible for a large number; and if this takes place before the brain is fully developed it will naturally tend to cause an arrest of development of certain parts, in addition to the actual destruction of nervous tissue produced by the lesion. Case No. 1 may well have been caused by occlusion of a basal perforating artery. Case No. 3 by occlusion of a branch of the sylvian artery (the lesion closely corresponds in its position with the softenings which are frequently found when one of the superior branches of this vessel is blocked). In case No. 2 the affected area almost exactly maps out the distribution of the great anastomotic vein which opens into the superior longitudinal sinus. The atrophy in this case was probably brought about by obstruction of this vein taking place during or shortly after birth; as a result, small necrotic foci occurred, followed later by sclerosis, with atrophy and distortion of the affected convolutions.

There are thus various conditions and many forms in which partial atrophy of one cerebral hemisphere may occur; but the effects resulting therefrom seem to vary considerably with the region of the hemisphere which is affected. In a large number of cases the lesion is confined to the cortex (as in cases 2 and 3), and in such there results a degeneration or arrest of development of the whole, or a portion, of one efferent tract; it is not usually complete, the pyramidal tracts in the medulla and cord still containing some healthy fibres. In such cases there is no change in the cerebellum, fillet, or the antero-lateral columns of the cord.

Several recorded cases of porencephaly instance this, as also do cases of long standing hemiplegia. As long as the lesion is confined to the cortex and does not involve the basal ganglia, the secondary effects are limited to the efferent tracts.

In those cases, however, in which the basal ganglia are involved, the effects are different; although in such the

efferent tract is usually affected also, its fibres being involved as they pass through the internal capsule. As a result of this there is atrophy of the white matter of the centrum ovale and of the larger cortical pyramidal cells, with dilatation of the lateral ventricle.

In addition to these changes there is pronounced atrophy in the middle fillet of the pons and medulla, and in the antero-lateral region of the cord on the *same* side as the lesion. There is also atrophy of the superior cerebellar peduncle, and the dentate nucleus, and cerebellar cortex of the *opposite* side.

Many cases of porencephaly, cerebral hemiatrophy, &c., have been already reported in which the basal ganglia were affected, and in most of them this atrophy of the fillet has been noticed, although no special attention seems to have been given to the point.

Such cases have been recorded by Wiglesworth and Campbell (25), Gibson and Turner (7), Norman and Fraser (19), Mahaim (12), Bruce (3), Spiller (23), and many others. In Norman and Fraser's case the interolivary layer on the affected side is said to differ little, if at all, from that on the healthy side; but a series of good photographs of the medulla, which accompany the paper, plainly show that it is but little more than half the size of the opposite healthy one.

Bruce attributed the change in the fillet in his case to atrophy of ascending fibres from the posterior column nuclei, and he states that he found a difference in the size of these nuclei on the two sides. This is the explanation which has generally been considered to account for the condition. One of us (F. W. M.) found atrophy of the posterior column nuclei after unilateral section of the fillet in the monkey (17). In our case, however, the absence of, or, at the most, slight change in the posterior column nuclei, and the atrophy of the antero-lateral column of the cord on the same side, make such an explanation not entirely satisfactory.

In none of the reported cases is there any mention of this atrophy of the antero-lateral column, indeed, in most such cases the cord does not seem to have been examined.

The atrophy in our case is quite definitely marked, and in the case of thrombosis of the middle cerebral artery to which we have referred, there is recent degeneration occupying an exactly similar position. It is, therefore, very desirable that more particular attention should be given to this point in the future examination of such cases, in order to see if the condition is constant in thalamic lesions.

The atrophy of the opposite cerebellar hemisphere in these cases is a well-known fact. It occurred in most of the above-mentioned cases; but we can find no record of the microscopical appearances of this change.

The fact that there is no change in the cerebellum in those cases in which the lesion is confined to the cerebral cortex, shows that there is no *direct* connection between these parts. In such cortical cases of long standing there may be secondary atrophy of portions of the thalamus, but this does not appear to lead to the same changes in the cerebellum and fillet as when the lesion is *primarily* basal.

Case 1 affords a very instructive example of secondary changes dependent upon an anatomical correlation of function, a lesion of the basal ganglia resulting in changes in brain, cerebellum and spinal cord.

Finally, it is very interesting to note that although in these cases the large motor cells of the cortex may be entirely absent, the tangential system of fibres is frequently but little affected. In case No. 2, in which there are practically no large pyramids, there is an abundant network of both tangential and inter-radial fibres, and this in a man who for thirty-three years had been subject to severe epileptic fits. A similar condition also exists in amyotrophic lateral sclerosis, and it may be said that it is only when symptoms of dementia become superadded that we are able to demonstrate atrophy of the tangential system of fibres.

Summary.

It will be well to briefly summarise the chief points of interest in connection with these cases:—

(1) In cases of cerebral hemiatrophy in which the lesion

is confined to the cortex there is sclerosis of the corresponding efferent tract throughout the pons, medulla and cord.

(2) In such cases in which the lesion primarily occurs in the basal ganglia, there is atrophy of the opposite half of the cerebellum with its superior peduncle, also of the mesial fillet in the pons and the interolivary layer in the medulla on the same side, in addition to the sclerosis in the efferent tract.

(3) Corresponding to the atrophy of the efferent tract, there is a numerical diminution in the anterior horn cells of the cervical and lumbar regions of the cord; such diminution being chiefly marked in the cells of the lateral and postero-external groups. In the same regions there is also a numerical diminution of the fibres composing the network within the anterior horns.

(4) As our cases show the cells of Clarke's column to be practically unchanged, the inference is that these cells do *not* form an inter-neuronic connection between the upper and lower efferent tracts.

(5) If the cortical lesion occurs at an early period there is a compensatory increase in the number of large motor cells of the opposite hemisphere.

(6) There may be pronounced atrophy of the fibres of the efferent projection system, and yet the association fibres remain unaltered. Not only may this occur in the brain, but also in the cerebellum.

(7) In the cerebellum there is a layer of granules lying immediately beneath the cells of Purkinje. These are distinct from the granules proper, they are somewhat larger, and are probably connected with an inter-cerebellar association system.

(8) There are fibres passing between the dentate nucleus of the cerebellum and the opposite basal ganglia.

(9) Two of the cases described in this paper point to the existence of a descending path having its origin in the thalamic region, passing thence through the middle fillet of the pons and the interolivary layer of the medulla to the antero-lateral column of the cord on the same side, and descending in this column as far as the lumbar region.

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DESCRIPTION OF THE PHOTOMICROGRAPHS.

FIG. 1.

Brain.—Section of the left occipital cortex stained by Weigert-Pal method. The occipital lobe is practically normal. The white and gray matter and the line of Gennari are well seen.

FIG. 2.

Brain.—Section from a convolution in the left central region, stained by Weigert-Pal method. The outer layer of cells and the line of Baillarger are practically normal. There are, however, no large pyramids, and there is a great diminution of all the nerve-cells below this layer. There are very few large fibres of the projection system in the medullary centre.

FIG. 3.

Cerebellum.—Section across a leaflet of the normal portion, stained by carmine-Weigert method, showing the outer molecular layer, the layer of Purkinje's cells, the inner layer of granules, and the medullary centre containing bundles of large fibres.

FIG. 4.

Cerebellum.—Section across a leaflet of the right lobus culminis, stained by Strœbe's method. This section shows an early stage of atrophy. The outer molecular layer is slightly thinner than normal. The majority of Purkinje's cells have disappeared, but two or three may still be seen. There is considerable diminution in the number of cells forming the inner granule layer, and owing to this, a narrow unchanged zone of outer granules is well seen forming a layer in the situation of the cells of Purkinje. The large fibres are practically absent from the medullary centre.

FIG. 5.

Cerebellum.—A leaflet from the same section as fig. 4, more highly magnified. This shows a still more advanced condition of the atrophy. There are no Purkinje's cells and very few of the inner granules. The layer of outer granules is on this account particularly well seen. There is an almost complete absence of large fibres from the medullary centre.

FIG. 6.

Cerebellum.—A leaflet from the right lobus culminis, stained by the carmine-Weigert method. This section is of the same magnification as figs. 3 and 4, and shows the full extent of the atrophy. The difference between it and fig. 3 is very striking. The outer molecular layer is very much narrowed, Purkinje's cells are entirely absent, and there are very few of the inner granules present. The layer of outer granules is particularly well seen. In the medullary centre there is an absence of large fibres.

FIG. 7.

Cerebellum.—A more highly magnified view of fig. 6. The small granules are seen to be very much diminished, and between this layer and the outer molecular layer are seen fine medullated fibres having a tangential course. There are no large fibres in the medullary centre, but a network of fine medullated fibres is here plainly visible.

FIG. 8.

Medulla.—Stained by Weigert-Pal's method, showing the atrophy of the left pyramid and interolivary lemniscus. There is also diminution of the left external and right internal arcuate fibres.

FIG. 9.

Spinal Cord.—Fifth cervical segment.

FIG. 10.

Spinal Cord.—Fifth dorsal segment.

FIG. 11.

Spinal Cord.—Tenth dorsal segment.

FIG. 12.

Spinal Cord.—Fourth lumbar segment.

All these sections of spinal cord are stained by the Weigert-Pal method. They show the marked asymmetry of the cord, the left half being considerably smaller. The atrophy of the left antero-lateral column, the huge size of the right direct pyramidal tract, the sclerosis in the right crossed pyramidal tract, and the other points mentioned in the text are well seen.

All the above sections are from Case 1.

FIG. 13.

Brain.—Section from the affected motor cortex of Case 2. Stained by Marchi-Pal method. This shows very well that the tangential, super and inter-radial fibres are present in abundance, although Nissl-stained sections show that there is an almost complete absence of the large pyramidal cells throughout the whole of the motor region of the same side.

FIG. 14.

Brain.—A more highly magnified view of a portion of fig. 13, showing an exceedingly well developed layer of tangential fibres.

A NOTE ON THE DEEP TRANSVERSE FIBRES OF THE PONS.

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THE lesion on which the following observations are founded was discovered while examining the nervous system of a patient who died from cerebral hæmorrhage. The patient was under the care of Dr. James Barr, at the Royal Infirmary Liverpool, and I am much indebted to him for permission to publish this note.

Although lesions of the pons are not very rare, yet an opportunity of examining the resulting degenerations is comparatively uncommon, and the injury in this instance is an unusually favourable one for the study of the course and relations of the deep transverse fibres.

The pons was hardened in Müller's fluid for a month, and thin slices treated with Marchi's fluid in the usual way. Sections were cut after paraffin embedding.

Lesion.—This is situated in the lower part of the upper half of the pons, about 2 mm. from the median raphe, and so placed as to intercept several large bundles of cross-fibres immediately posterior to the pyramidal tract, and, indeed, to involve to a small extent two bundles of the latter tract. In cross-section it is of an irregularly oval shape, with its long diameter, about 3 mm. in length, transverse. It extends about 2 mm. from above downwards.

It is apparently a softened patch, due most probably to thrombosis of some of the small vessels. There are no signs of hæmorrhage in it, old or recent. Microscopical examination of the softened area shows that it consists of a compact mass of leucocytes, traversed by one or two

capillaries; these, as well as the small surrounding vessels, being full of blood. The leucocytes are large, and accompanied by numerous giant-cells, both varieties being filled with granules blackened by the osmic acid.

The lesion is strictly unilateral and does not approach the middle line nearer than 2 mm. at any point, and is, I think, unusually interesting on account of its circumscribed character.

Transverse fibres.—These are degenerated on both sides

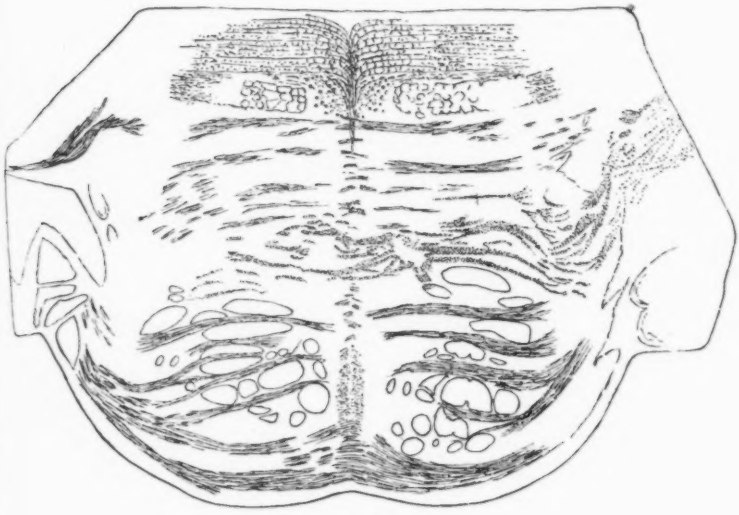

Fig. 2.

of the pons, but the altered fibres are much more numerous on the side of the lesion than in the opposite half.

A section at the level where the softened area is greatest shows the following features (fig. 2):—(1) On the outer side of the lesion there is heavy degeneration in three main bundles of the cross-fibres, in several minor bundles in the immediate neighbourhood, and also in a thin transverse tract considerably posterior to the main part. Following these tracts laterally, they are seen to break up into smaller

bundles, extending over a large area, in the region of the lateral gray matter, and here apparently many of them end. Large numbers of fibres, however, pass beyond the gray matter, and enter in a more or less scattered fashion into the middle cerebellar peduncle, occupying mainly its inner half.

There is also a small degeneration in two of the posterior bundles of the pyramidal tract.

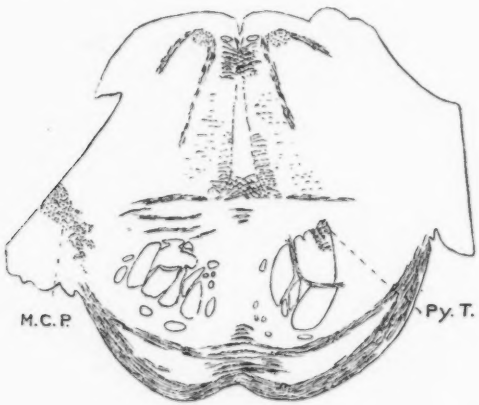

FIG. 1.

Section through the lower limit of pons.

M.C.P.—Degenerated tract in the middle cerebellar peduncle.
Py.T.—Degenerated fibres of the pyramidal tract.

Section at the level of the lesion. The main features are drawn carefully to a magnification of four diameters, so that the area over which the degeneration spreads is accurately represented.

(2) On the inner side of the lesion there is degeneration, also in three bundles. These pass directly to the median raphe, where they cross and have the same course as the above. A few scattered small bundles are seen in the neighbourhood of the raphe some little distance posterior to the lesion. The section from which the figure in the plate was drawn is slightly oblique, and consequently the mode of termination of these fibres on this side is not

shown, but in all respects it is the same as on the side of the lesion.

Sections somewhat above or below this level show the same general appearances, but the number of degenerate fibres diminishes, of course, with the area of softening.

Fig. 1 represents a section through the pons at its lower limit, below the injury. The only descending degeneration is in the posterior part of the pyramidal tract. This is small, and due to the fact above-mentioned, that the softening encroached slightly upon two small bundles of the aforesaid tract. The further course of this degeneration I had not the opportunity to follow. On the opposite side there are still seen some of the cross-fibres remaining, most of which are passing into the middle cerebellar peduncle. The degeneration remaining in the other middle peduncle at this level is too scattered to be represented in the figure.

Sections above the lesion show no degeneration in the fillet or the posterior longitudinal bundle, nor are there any fibres ascending to the corpora quadrigemina.

By studying sections immediately above and below the lesion it is seen that the degenerated transverse fibres are practically confined to the level of the injury, this being an indication that they maintain a fairly horizontal course until they reach the lateral limit of the pons, where, of course, they dip backwards somewhat with the middle cerebellar peduncle.

The lesion from its nature and situation cannot throw much light on the origin of these fibres, but some interesting points as to their destination may be ascertained. Pellizi (1) finds that destruction of the middle lobe of the cerebellum causes degeneration in the median field of the middle peduncle and in the stratum profundum of the pons. According to Cajal ponto-cerebellar fibres spring from the cells of the pontine nuclei. These cross the middle line and pass to the cerebellum by way of the middle peduncle of the opposite side. This is in all probability the origin of many of the degenerated fibres in the present case.

The exact constitution of the middle cerebellar peduncle

is not by any means a settled question. Marchi, and Ferrier and Turner found, after destruction of one half of the cerebellum, complete atrophy of the middle peduncle and of all the cross fibres of the pons. Risien Russell states that the latter fibres pass to the pons ganglia of the opposite side.

On the other hand, according to Thomas (2), the above lesion entails degeneration of only a small portion of this peduncle, and he concludes that its fibres are passing from the pons to the cerebellum. Klimow (3) similarly comes to the conclusion that this peduncle consists largely, if not exclusively, of centripetal fibres, originating in a great measure from the pons gray matter of the opposite side with a termination in the flocculus and hemisphere.

The degenerated fibres in the middle peduncle in the present case are, of course, centripetal, and although they are scattered in each section, yet from the depth through which they extend one must conclude that there is a very considerable number of them.

Inasmuch as the lesion is so near the middle line, one may conclude that all the damaged fibres of the same side have really crossed. Nevertheless, one is unable definitely to decide upon the origin of these fibres, which is in all probability a multiple one. Their termination, however, is definitely twofold, namely, (1) in the pons ganglia; (2) in the cerebellum.

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TWO CASES OF ARRESTED DEVELOPMENT OF THE NERVOUS SYSTEM IN CHILDREN.

BY FREDERICK E. BATTEN, M.D.

(1) Arrested development of the left cerebral hemisphere and corresponding pyramid.

(2) Arrested development of the cerebellum with hydromyelia.

Case 1.—A male child, aged six months, was admitted to the Hospital for Sick Children under Dr. Lees, and to him my thanks are due for permission to make use of the clinical notes. When a week old twitching of the left hand and foot were first noticed, when four months old both sides of the body became affected, and for the last fourteen days the child had "gone purple" in the fits. It was the fifth child, all the others being healthy. It was born at the eighth month, and the labour was natural. No evidence of syphilis could be obtained. Its parents were living and healthy.

On admission the child was ill-nourished, weighing 6½ lbs., with a narrow forehead and a markedly asymmetrical head and face, the right side being larger than the left. There was, however, ptosis of the left eyelid, and at the same time marked prominence of this eye. The orbicularis palpebrarum acted well.

There was congenital absence of the left thumb.

The trunk and limbs of the child appeared symmetrical, the right arm-jerk was more active than the left, the legs were rigid and the knee-jerks active and equal; no ankle clonus. There was no paralysis of the limbs.

The child had frequent fits, which always terminated by the child sneezing (according to the statement of the sister). During the fits the twitching began on the left side of the face, the eyes were turned to the right, the left leg drawn up, and the left arm flexed, the right leg rigidly extended. The child died a few days after admission to the hospital.

Post mortem.

The asymmetry of the skull was very marked, the prominence of the left eye noted during life being due to the position of the orbital plate.

The left hemisphere of the brain was very much smaller than the right, its substance being hard and firm, the convolutions remaining at the normal development usually reached between the sixth and the seventh month of fœtal life.[1] The right side of the brain was normally convoluted. There was obvious deficiency on the left side of the pons and medulla. The cerebellum

	Right Hemisphere.	*Left Hemisphere.*
Length ..	11·5 cm. ..	10 cm.
Weight ..	5¾ oz. ..	3 oz.

was noted as being abnormally large as compared to the size of the brain, being almost as large as that of a child of fifteen months, but there was apparently no asymmetry of the lobes. (No microscopic examination of the organ was made.) All the cranial nerves were normally situated with the exception of the first on the left side, and this may have been torn during the removal of the brain. The eyes were natural, and all the other organs of the body were healthy.

Microscopical examination.—The motor region of the cortex, both on the right and left sides, was examined by Nissl's method. There would seem to be a diminution of the pyramidal cells on both sides, but this diminution was more marked on the left than on the right; the left cortex, however, contained some abnormally large pyramidal cells.

Sections at the level of the corpora quadrigemina showed that the left crus was very considerably smaller than the right. The decussation of the superior cerebellar peduncle was present, and the fibres appeared more abundant on the left side. The posterior longitudinal bundle was well seen and was symmetrical, and the descending root of the fifth was better developed on the right side than the left.

Sections through the pons showed some very marked features; there was almost complete absence of the pyramid on the left side, the fillet on the left side was likewise almost absent, represented only by a very small bundle of fibres, and the superior peduncle of the cerebellum of the right side was very much diminished in size, the left being normal in appearance. The posterior longitu-

[1] Retzius. *Das Menschenhirn*, tafe xxviii.

dinal bundles appeared symmetrical, and the transverse fibres of the pons also appeared about equal on the two sides.

Sections at the level of the eighth nerve showed that the left pyramid was absent, the left fillet was absent, the ascending root of the fifth nerve was equal on the two sides, but the transverse fibres of the pons appeared at this level to be more abundant on the right than the left. This may, however, have been due to the line of the section being somewhat oblique.

At the level of the olive the inferior peduncles of the cerebellum were equally developed, the olivary bodies on the two sides were practically symmetrical, although the right may have been somewhat larger than the left.

At the level of the decussation of the fillet, except for the fact that there was only one pyramid, no other abnormality could be detected. The funiculi gracilis and cuneatus were equal on the two sides.

Spinal Cord.

Cervical region.—The cord to the naked eye appeared almost symmetrical; there was, however, a slight diminution of the white matter on the right side, and under the microscope there was an obvious lack of fibres not only in the crossed but also in the direct pyramidal tract. The central canal in one of the sections was double, and in another was dilated. The grey matter and the cells of the anterior horn appeared normal.

Dorsal region.—The cord in this region appeared normal, except for a slight difference in the size of the white matter on the two sides.

Lumbar region.—The cord in this region appeared perfectly normal.

Remarks.

One of the most interesting features of the case lies in the fact that the child was not hemiplegic in spite of the non-development of one hemisphere and one pyramid, but the condition found in the cord below the level of the decussation gives the explanation, since both sides of the cord would appear to be innervated from one hemisphere. This has previously been shown to be the case by Russell in a cat, in whom, although both hemispheres were normally developed, there was absence of the right pyramid and the

right lobe of the cerebellum. The cord, however, below the decussation appeared normal, and he showed that at the decussation of the pyramid the fibres bifurcated, one set (the larger) passing to the pyramidal tract of the opposite side, while the lesser bundle passed to the pyramid on the same side.

In Russell's case the atrophy of the fillet, superior, middle, and inferior cerebellar peduncles and the pyramid were on the same side. In the present case the pyramid and fillet were atrophied on the same side and the superior cerebellar peduncle on the opposite side, the middle and inferior peduncles being normal. In his case the opposite inferior olive and funiculus cuneatus were atrophied, while in the present case they were apparently symmetrical and normal.

Wiglesworth has also described two cases of porencephaly of one hemisphere with absence of the corresponding pyramid, but in his cases the condition was associated with hemiplegia and descending degeneration in the cord.

Case 2. *Arrest of Development of the Cerebellum with Encephalocele and Hydromyelia.*

A female child, aged 4 weeks, was admitted to the hospital for Sick Children under Mr. Pitts (to whom I am indebted for the clinical notes) with an occipital encephalocele, which was present at birth, but gradually increased during the first weeks of life. The sac was translucent, and measured $4\frac{1}{2}$ in. round the base. The tumour was removed, and the child did well for eight days, when it died somewhat suddenly.

Post mortem, made twenty-four hours after death. It was found that the meninges passed through a small round hole just large enough to admit the little finger in the occipital bone to form the sac of the meningocele. The hole in the bone was situated in the midline immediately below the torculus Herophili, and it was thus just below the tentorium. The substance of the cerebellum also tapered off, and passed just through the hole in the bone, but probably not further into the sac. The shape of the cerebellum was roughly pear-shaped. The lateral lobes of the cere-

PLATE I.

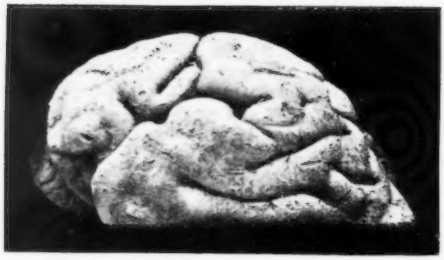


FIG. 1.

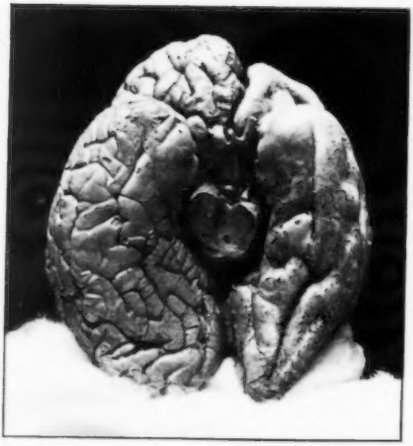


FIG. 2.

bellum were much smaller than normal, and there was some sclerosis in parts. The right lobe appeared rather smaller than the left. The cerebrum was normal to the naked eye, no abnormality, congenital or otherwise, could be detected. The posterior fossa of the skull was curiously deformed, the part below the tentorium being very small and pear-shaped, corresponding to the defective development of the cerebellum.

On the more detailed examination of the cerebellum it was found that the stalk which passed into the cyst was formed of two canals. On passing a probe into the upper one it was found to pass towards the iter, while into the lower one the probe passes into the fourth ventricle. On opening up the canal, however, it is found that they are only separated from one another for a short distance from the point where they are cut off, and both entered the space between the lobes of the cerebellum. The superior peduncle of the cerebellum, which was much smaller than normal, passed back on the dorsal surface of the cerebellum, the lobes of the cerebellum that existed lying wholly ventral to these peduncles. The middle peduncles were normal in situation and in appearance. The inferior peduncle is represented by a small filament which passed up into the wall of the cyst. The two lateral lobes of the cerebellum were completely separated, and it was only the most ventral portion of them that appeared developed. The middle lobe was absent.

Spinal Cord.

The spinal cord at the level of the second cervical was cleft on the dorsal surface, so that the posterior columns were separated from one another by a cavity that was continuous with the central canal of the spinal cord. This cleft extended from the second cervical to the eighth cervical, at which level the cord appeared normal. At the level of the third dorsal, however, a cavity appeared in the centre of the cord, being a dilatation of the central canal. This extended as far as the ninth dorsal, but the greatest amount of dilatation occurred at the level of the fifth dorsal. The lumbar cord appeared normal. The staining was carried out by the Heller-Robertson method. The microscopical examination did not add much to one's knowledge; it proved, however, that the spaces in the cord were true dilatation of the central canal, since they were lined with columnar epithelium. The following is a description of the cord at the various levels:—

Section just anterior to the pons.—At this region the decussa-

tion of the superior cerebellar peduncles, the fillet, and posterior longitudinal bundle, appeared normal; the pyramidal tract seen on the ventral side of the section was not well stained.

Section through the cerebellum and pons.—A transverse section was made through the cerebellum and pons. The complete separation of the two lobes of the cerebellum was a very noticeable feature. There was obvious sclerosis of the cerebellar substance, especially on the right side. The fillet, the posterior longitudinal bundles, and the pyramidal tract were normally developed, and the fibres from the inferior cerebellar peduncle were well stained; the transverse fibres of the pons were but little stained.

Section through the medulla in the region of the olive.—The olive was fairly well developed on either side, the interolivary stratum was well stained, the fibres of the inferior peduncle and the ascending root of the fifth were apparently normal. The vagus nerve can be seen normally stained.

Section through the decussation of the pyramid.—At this region the only abnormal feature was the fact that the dorsal median fissure was continuous with the central canal of the spinal cord.

Section at the level of the fourth cervical.—At this level the cord was normal, except for the fact that there was complete separation of the posterior columns, and the dorsal median fissure was continuous with the central canal; the space was lined by epithelium, which proved it to be a true dilatation of the central canal. The tracts and cells of the cord were normal.

At the level of the eighth cervical.—At this level there was no longer a separation of the posterior columns, but there was an abnormal amount of neuroglia tissue situated in the position of Goll's column. The central canal at this region was larger than normal, and had a handle-like projection into the neuroglia tissue in the posterior region.

At the level of the third dorsal.—A triangular-shaped cavity existed, the posterior columns forming a horse-shoe round the neuroglia.

At the level of the seventh dorsal.—The central canal was much dilated, and the posterior columns formed a horse-shoe round the neuroglia tissue, which was present on the dorsal side of the central canal.

At the level of the first lumbar.—At this level the cord has assumed its normal appearance.

PLATE II.

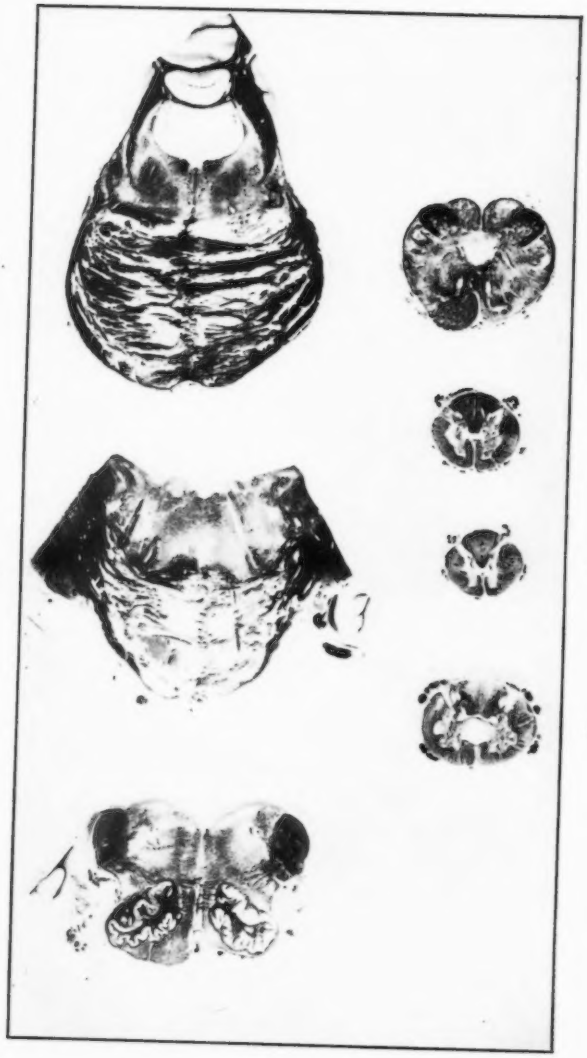


Fig. 1.

Fig. 4.

Fig. 7.

Fig. 2.

Fig. 6.

Fig. 5.

Fig. 3.

PLATE III.

Remarks.

The abnormality met with in this case would seem to be that which is usually found in cases of occipital meningocele, *i.e.*, lack of development of the cerebellum, the cyst communicating with the fourth ventricle. Two canals, as in the above case, have been described as passing through the opening in the skull.

A similar case was described by Sutton in his "Tumours," and the specimen is said to be in the museum of the Middlesex Hospital. In this case there was said to be no cerebellum, but the flocculus was large and conspicuous. He also points out that the flocculus is developed from lateral outgrowths from the floor of the third vesicle, while the cerebellum is developed from the foremost part of the roof of that vesicle.

Leyden also described two cases of meningocele in the occipital region, with absence of the cerebellum and dilitation of the central canal in the cervical and dorsal regions, the first case being that in a child two and a half years old and the second in a child two years old.

REFERENCES.

(1) Russell. Brain, vol. xviii., p. 37, 1895.
(2) Wiglesworth. Brain, vol. xx., p. 88, 1897.
(3) Sutton. "Tumours," p. 452.
(4) Leyden. *Virchow's Archiv*, Bd. lxviii., plate I., p. 1.

DESCRIPTION OF PLATES.

Plate I.

Fig. 1.—Lateral view of left cerebral hemisphere of child, aged 6 months, showing arrest of development at about the sixth to seventh month of fœtal life. (Comp. Retzius, *Das Menschenhirn*, tafel xxviii.

Fig. 2.—Ventral aspect of the same brain, showing the marked asymmetry between the two hemispheres, and also the lack of development of the left crus.

Plate II.

Fig. 1.—Section through pons of same case, showing absence of left pyramid, left fillet, and diminution in size of right superior cerebellar peduncle.

FIG. 2.—Section at the level of the eighth nerve, showing absence of left pyramid and left fillet.

FIG. 3.—Section at the level of the olive, showing the absence of left pyramid, the symmetry of the olives and of the inferior peduncles of the cerebellum.

FIG. 4.—Section at the level of the decussation of the fillet, showing the absence of the left pyramid and the symmetry of the funiculi gracilis and cuneatus.

FIG. 5.—Section of cord in cervical region, showing that the right side of the cord is smaller than the left, and there is some dilatation of the central canal; it can also be seen that the fibres in the direct pyramidal tract of the left side and the crossed pyramidal tract of the right side are not medullated.

FIG. 6.—Section of cord in dorsal region; the asymmetry is less marked than in the cervical.

FIG. 7.—Section of cord in lumbar region; the asymmetry of the cord is very slight, the right side being slightly smaller than the left.

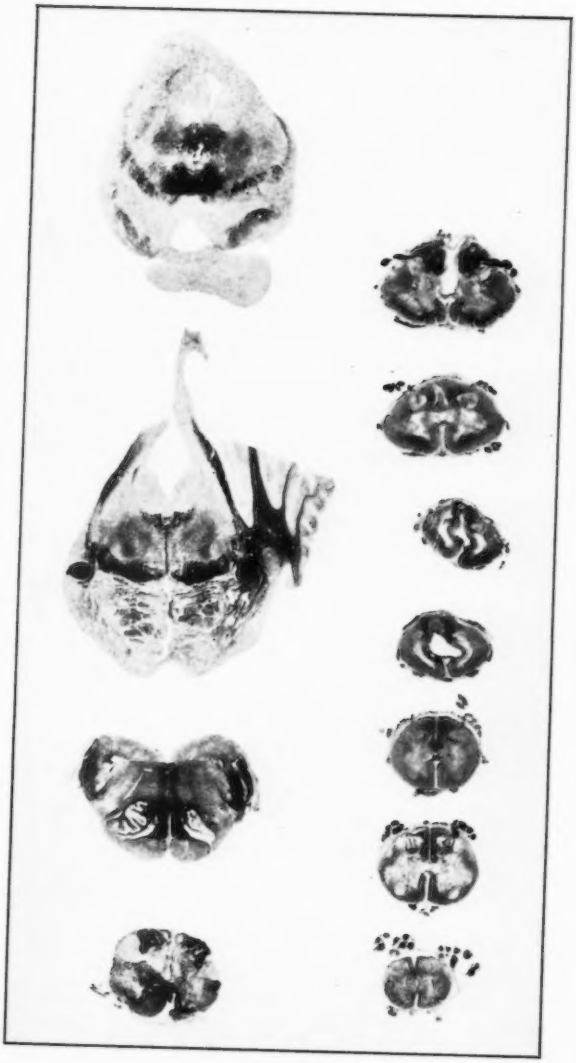
PLATE III.

Section through pons and cerebellum of child, aged 4 weeks, with occipital encephalocele, showing complete separation of the lobes of the cerebellum, with sclerosis of the right. The middle lobe is absent. (The cut separating the right lobe of the cerebellum from the pons is artificial.) The cavity between the two lobes communicated with the cyst in the occipital region.

PLATE IV.

Sections at various levels through the pons, medulla, and cord, showing the separation of the posterior columns in the cervical region and the dilatation of the central canal in the dorsal region, and the normal appearance of the cord in the lumbar and sacral region.

PLATE IV.



Critical Digest.

MYASTHENIA GRAVIS.

BY

HARRY CAMPBELL, M.D., F.R.C.P.

AND

EDWIN BRAMWELL, M.B., M.R.C.P.

THE disease known as myasthenia gravis has hitherto attracted little attention in this country. Nevertheless some sixty cases have been recorded within the last few years, chiefly by German writers, and the disease has found a recognised place in German text-books on neurology.

We have ourselves had the opportunity of examining nine cases.

The disease has received various names. The earlier cases were published as "cases of bulbar paralysis without discoverable anatomical changes," and some recent writers have adopted this nomenclature. Strümpell introduced the term "Asthenic bulbar palsy." The affection is, however, not confined to the bulbar muscles. Other names which have been employed are "general profound myasthenia," "Erb's disease," the "Erb-Goldflam" and the "Hoppe-Goldflam-symptom-complex." Jolly has proposed the term "myasthenia gravis pseudo-paralytica," and this, or for short "myasthenia gravis," appears to us the most suitable and convenient hitherto suggested.

SALIENT FEATURES OF THE DISEASE.

There is weakness, sometimes amounting to complete paralysis, of some or all of the voluntary muscles. After prolonged rest of the affected muscles, *e.g.*, the first thing in the morning, they may respond normally to the will, but

they become rapidly exhausted after voluntary contractions, regaining their power again after rest. In severe cases, however, weakness and, indeed, actual paralysis may persist even after prolonged rest.

The affected muscles often exhibit the "myasthenic reaction," becoming exhausted by faradic stimulation just as they are by voluntary effort; galvanism, on the other hand, having little power in this respect.

The entire system of voluntary muscles may be affected, but those muscles are most apt to be implicated which normally act most constantly, such as the cervical muscles and the extrinsic muscles of the eye-balls. The bulbar muscles are very generally involved. Hence the term "asthenic bulbar paralysis."

A characteristic feature of the disease is its tendency to fluctuate in severity from day to day, or from week to week, or even to disappear for months or years, to reappear.

There are no sensory symptoms. Death occurs in a large proportion of the cases, but no structural changes have been discovered to account for the symptoms.

Historical.

Wilks (1), in a paper published in *Guy's Hospital Reports* for 1877, describes a case of bulbar paralysis in which no anatomical changes were found after death. This is the first reference to myasthenia gravis to be found in the literature.

Erb (2), in the following year, described a peculiar form of bulbar disease ("einen eigenartigen wahrscheinlich bulbären symptomen complex"), the characteristic symptoms of which were: ptosis, weakness of the jaws and neck muscles; and of secondary importance—weakness of the tongue and extremities, difficulty in swallowing, and weakness of the upper part of the face.

The publications of Wilks and Erb do not appear to have attracted much attention, for no further publication followed until 1887, when two cases were recorded, the one by Oppenheim (5), the other by Eisenlohr (4). In 1890 two cases were described by Bernhardt (7) and Leonard Shaw

(6), but it was not until the papers of Jolly (8), Hoppe (11), and Goldflam (9) appeared, that the disease was established as a definite clinical entity.

Hoppe, in 1892, was the first writer to give a full account of the disorder.

In 1895 Strümpell* reviewed the literature of the disease and collected twenty undoubted examples which had been reported up to that time. In 1898 an excellent *resumé* of the subject appeared by Laquer (40) of Frankfort.

During the last four or five years there have been numerous additions to the literature of the disease, and there are now some sixty or seventy cases on record, ten having been described in this country. †

The most complete account of the disease published in this country is that of Buzzard (55).

Of the cases hitherto recorded ten have been in England, four in America, eight in France, five in Italy, the remainder in Germany and Austria.

It is difficult at present to form an idea as to the frequency of a disease the clinical picture of which is so little known to the medical profession. The condition is undoubtedly a rare one. Erb did not meet with another case for twenty years after his original description.

During the last six months four patients suffering from myasthenia gravis have been treated in the National Hospital, Queen Square.

Etiology.

Males and females are almost equally affected. The age at which the sexes are attacked is different. Of fifty-eight cases the average age was for men 35 and for women 24 years. The youngest patient in whom the disease has been

* Strumpell's cases:—

Wilks (1)	Erb (3)	Eisenlohr (1)
Oppenheim (1)	Shaw (1)	Bernhardt (1)
Jolly (2)	Goldflam (4)	Hoppe (1)
Reinak (1)	Pineles (3)	Strümpell (1)

† Wilks (1) Shaw (1) Dreschfeld (1)
Suckling (2) Clifford Allbutt (1) Guthrie (1)
Harry Campbell (1) Buzzard (2)

observed—excluding a doubtful case in a child aged 2½ described by Mailhouse, (39)—was 12, the oldest patient, 55.

With regard to occupation, no conclusive deductions are to be drawn, although in the majority of instances in which mention of this point has been made the patients have been manual workers.

Rarely has there been a family tendency to nervous disease. Exceptions to this are found in cases recorded by Goldflam (9), Sinkler (50), Buzzard (55), Suckling (15), and Kojewnikov (30). No case is on record in which more than one member of a family has been affected. As frequently as not the malady attacks individuals who have previously enjoyed good health. A considerable percentage of the female patients have been anæmic. Laquer's patient suffered from attacks of gout. In a few instances there have been signs of a defective nervous system. In Berkeley's (34) case there was an obvious mental defect. In a case described by Karplus (37) the patient had recurring ptosis from the time she was 5 years of age. In Saenger's (43) case the patient had infantile paralysis when a child. Eisenlohr's patient as a child suffered from megrim occasionally accompanied by ptosis. With these exceptions there is a striking absence of a nervous or hysterical temperament.

Syphilis and alcohol appear to play no part as causative factors.

Not infrequently the symptoms have followed upon some previous affection, such as influenza (Remak, 12), scarlatina (Campbell, 53), typhoid fever (Sinkler, 50), sore throat (Saenger, 43; Eulenberg, 38), otitis media (Jolly, 20), an attack of diarrhœa (Shaw, 6), and acute intestinal obstruction (Feinberg, 57). In one case the onset of menstruation (Kojewnikov, 30), in others a confinement (Punton, 51, Laquer, 40), appear to have acted as exciting causes. Emotional excitement (Angelini, 49), a chill (Kojewnikov, 30; Clifford Allbutt, 53), the extraction of a tooth (Wheaton, 41) and an attack of megrim (Eisenlohr, 4) have been blamed. In two cases the illness was attributed to taking cold baths (Buzzard, 55; Montesano, 45), in others to over-exertion (Suckling, 15, Buzzard, 55).

Mode of Onset.

The disease usually comes on gradually, although in a few cases the onset has been sudden. Occasionally slight headache, pain in the neck and back, giddiness and photophobia have immediately preceded the first symptoms of motor weakness.

Most frequently the characteristic weakness first shows itself in the muscles innervated by the bulb. Thus, in the case of a schoolboy, the first symptom was an inability to keep the eyes open—he was unable to look at the blackboard on account of the dropping of the upper eyelids. Diplopia, weakness of the lips, indistinct speech, difficulty in masticating and in swallowing, and inability to hold the head erect, have each been met with as the earliest manifestations of the disease. A considerable number of cases have been described in which the limbs were first affected. An hotel cook found that his fingers were weak after rolling dough. Another patient first noticed weakness in the fingers after sawing wood. A housemaid, who had a great deal of "stair work," became very weak in the legs, so much so that on several occasions the legs gave way and she fell to the ground. In some cases the initial weakness is more general, affecting both the limbs and bulbar muscles.

Symptomatology.

Bulbar muscles.—Ptosis is a common symptom; it is usually bilateral and often more marked on one side than the other. Sometimes the ptosis is constant; in other cases it is only present towards the latter part of the day, or if the patient attempts to look up for any length of time, when the upper lids gradually fall. On account of weakness of the occipito-frontales muscles, frontal overaction, which is so frequently associated with the ptosis of tabes and serves to antagonise the drooping of the lids, cannot occur. The only way in which the patient can counteract the ptosis is by throwing back his head, but here again weakness of the neck muscles makes it difficult for him to maintain this position.

Some interesting observations in connection with ptosis were made in Dr. Buzzard's male case. It was observed by Mr. Marcus Gunn that on appealing to the patient's emotions and occasionally upon mental concentration, the upper lids were temporarily raised to some extent, in association with a certain amount of dilatation of the pupils. In the same patient on another occasion, just after he had awakened from sleep, the upper lids were retracted so that the sclerotic was visible above the corneal margin. In this, as in several other recorded cases, von Graefe's sign was at times present. These facts would suggest that the fibres of Müller are not affected.

Weakness of the external ocular muscles with consequent diplopia is often present. Sometimes one muscle is more affected than others, sometimes there is a general paresis affecting all the extrinsic muscles, while in some cases complete and persistent ophthalmoplegia externa has been present. A striking feature of the diplopia in myasthenia gravis is the alteration in the relative position of the two images at different times.

In some cases irregular nystagmoid movements, similar to those seen in conditions of general asthenia, are induced upon conjugate lateral deviation of the eyes. The ocular muscles, as is the case with other voluntary muscles, become readily fatigued; patients frequently complain that after reading a few lines the letters and words become blurred and indistinct, and run into each other.

Pupil changes are quite exceptional. The pupils are sometimes unequal. In one case, recorded by Brissaud and Lantzenberg (36), in which, however, the diagnosis appears doubtful, they did not react either to light or accommodation, while in one or two others the reactions were sluggish.

In Dr. Buzzard's case, already alluded to, after prolonged convergence the pupils showed a great tendency to oscillatory movements. Attempts to tire out accommodation in this case, as in others in which the experiment has been tried, were unsuccessful, nor was it found possible to exhaust the sphincter iridis by holding a bright light in front of the eyes.

Difficulty in masticating is one of the most constant symptoms of the disease. The difficulty may be slight and only noticeable after a meal, and more especially towards evening. Sometimes the weakness of the muscles of mastication is so great that the finger may be placed with impunity between the patient's teeth while he closes the jaws with all the force he can command. Often the mouth is kept slightly open because of the weakness of the elevators of the jaw. In cases in which the weakness of the muscles of mastication is great, there is a tendency for the lower jaw to drop, which causes great distress to the patient. In several cases the patient was in the habit of supporting the lower jaw with the hand while masticating.

In Buzzard's first case a curious symptom, connected with the jaw muscles, was complained of. The patient stated that not infrequently he was wakened from sleep by a champing movement of the jaws, which sometimes resulted in his biting his tongue. This symptom was probably due to a clonic contraction of the elevators of the jaw, and is comparable with the clonic movements which have been occasionally noticed in the extremities. Patients sometimes complain of an increased flow of saliva, probably comparable to the well-known dribbling of bulbar paralysis.

The facial appearance is striking and characteristic. The immobile, expressionless face and the drooping upper lids remind one of the Landouzy-Dejerine form of myopathy. Often there is complete inability to wrinkle the forehead, raise the eyebrows, or to frown. Erb (2), in describing his early cases, draws attention to this upper facial weakness.

The sphincters of the orbit are often so weak that the patient is unable to keep his eyes closed against the slightest resistance. In many cases there is great weakness in the muscles about the mouth. A very poor attempt is made to raise the upper lip and show the teeth; the patient cannot pout the lips, "empty a spoon with the upper lip," whistle, or blow out a candle. In extreme cases fluids dribble from the mouth while drinking. In one case the patient was in the habit of supporting the lower lip with her fingers to prevent this occurring.

Difficulty in swallowing is another symptom which is rarely absent. In several instances death has followed attacks of choking brought on while swallowing. Fluids are, as a rule, more easily taken than solids, although, on account of weakness of the palate, they frequently regurgitate through the nose. One patient, when swallowing, always supported the hyoid bone with her hand. The pharyngeal reflex is often absent, a fact which is probably of importance in explaining this symptom.

Weakness of the palate is of frequent occurrence, the voice becomes nasal, and, if the weakness is marked, fluids regurgitate through the nose when the patient drinks. The patient is unable to blow out his cheeks, the air escaping through the nose. The movement of the palate is almost always impaired and becomes much more feeble after repeated vocalisation of the letter ā.

The palatal reflex is usually diminished, and may be absent.

Paralysis of the laryngeal muscles has been but rarely noticed. In one case there was paralysis of one vocal cord (Punton, 51); in another movement of one cord was defective (Bernhardt, 7). In other cases the tone of the voice has been lowered, suggesting weakness of the adductors.

In Hoppe's case there was weakness of both abductors and adductors. In one case (Dreschfeld, 14) the abductors, in another the adductors were weak (Kojewnikov, 30). In Dr. Buzzard's first case it was remarked by Sir Felix Semon that after repeated deep respirations the cords did not separate so much as at first, clearly showing myasthenic weakness of the abductors. In two cases, those of Wernicke (16) and Toby Cohn (31), anæsthesia of both pharynx and larynx was present.

A feeling of fatigue and of "aching" and "stiffness" in the tongue is often complained of, especially after speaking or eating. The movements of the tongue may be impaired. The patient can usually protrude the tongue, but cannot long maintain it in this position, and he may not be able to thrust out the cheek forcibly with it.

Very characteristic alterations in speech occur. It may

be that the patient speaks naturally when he begins, but soon speech becomes nasal; words become more and more indistinct; the voice becomes weaker and weaker, and finally he has to stop for want of breath. The altered speech is due, in part, to weakness of the articulatory apparatus—lips, jaws, tongue, and soft palate; in part to defective action of the respiratory muscles, which make it impossible for the patient to take in sufficient air for phonation. These peculiarities are well demonstrated when the patient reads aloud.

Trunk muscles.—The myasthenia, as already observed, involves the muscles of the trunk and limbs, as well as those just considered.

Weakness in the neck muscles and consequent inability to support the head is common. In some cases the head tends to fall forwards, in others backwards, and it often requires support.

The muscles of the back may suffer in a similar manner, so that the patient cannot sit up for any length of time without feeling tired. On account of weakness of the abdominal muscles he may be quite unable to raise himself from the recumbent to the sitting posture without using his hands. So marked is the weakness of the trunk and limb muscles in some cases that there is the greatest difficulty in turning in bed.

The muscles of respiration may likewise be involved. In one case the difference between the circumference of the chest at full inspiration and complete expiration amounted to a quarter of an inch only. The dyspnœa, which results on even slight exertion, finds a ready explanation in this fact. Besides the dyspnœa which follows exertion, unaccountable attacks of breathlessness occur, during which the patient is in danger of his life. Strümpell (24) gives a graphic account of the dyspnœic attacks which occurred in his case:—"The patient falls to the ground if walking, becomes cyanosed and breathless. Respiration is noisy and restless; sputum accumulates in the mouth; he can neither swallow it nor spit it out. The tongue appears to sink back into the mouth. If in bed he sits up with head bent forwards, the saliva flowing from his mouth. The

arms when raised fall flaccid to the side." These attacks may persist for several hours. Strümpell believes that they are due to sinking back of the tongue, and in his case he was in the habit of pulling the tongue forward with marked benefit. Attacks of palpitation are not uncommonly complained of, sometimes in association with dyspnœa, at other times quite independently. The palpitation may be entirely subjective and unassociated with any increase in the pulse rate.

Extremities.—It has been pointed out that in the extremities the muscles situated near the trunk usually suffer most. Not infrequently the first weakness in the arms is noticed when the patient undresses at night to go to bed. One of the earliest symptoms in the case of a female patient was inability to raise the arms in order to brush her hair. But the weakness is by no means confined to the shoulder and upper arm muscles. In two cases, already referred to, weakness in the fingers was the first symptom to appear. The handwriting may show characteristic changes dependent upon the myasthenic condition. At first the letters are well formed and the patient writes fairly rapidly, but gradually the writing becomes slower and more laboured and the letters badly formed, until finally he has to give up the attempt. Weakness in the arms may be so great that the patient cannot feed himself. The ilio-psoas and quadriceps would seem to be the muscles which are as a rule most involved in the legs. As a rule the patient is unable to walk a long distance, sometimes he cannot walk fifty yards, indeed, he may not be able to walk at all.

In slight cases the patient appears to walk quite naturally. When the legs feel tired he rests for a minute or two, and then walks on almost as well as before. In cases in which the lower extremities are very weak the gait is waddling in character.

Sudden giving way of the legs is a not infrequent symptom, and was first drawn attention to by Strümpell.

Factors influencing the myasthenic condition.—Emotional excitement increases the intensity of the symptoms in a very striking way. In one of Goldflam's cases a relapse

occurred after a fright. The presence of a strange doctor may be sufficient to intensify the symptoms for the time being. In one case reading an exciting description of a battle produced a similar result.

Buzzard's patient always felt worse in cold weather. In a case under the care of Dr. Ormerod five years ago, which presented many of the features of myasthenia gravis affecting the limbs, the patient, a blacksmith, writes to say that he is now quite well, although in the cold weather he still notices some weakness in his fingers. The myasthenia in Campbell's second case was always increased by cold weather.

Female patients are often worse at the menstrual periods. Such was very decidedly the case in one of our own patients. As we have seen, several of the recorded cases have come on after a confinement.

The speedy production of muscular exhaustion constitutes the most striking feature of the disease. Persistence in a movement causes it to become gradually weaker, and ultimately impossible, until after a short rest, when it can again be repeated.

As Strümpell pointed out, persistent paresis picks out especially those muscles whose function necessitates persistent activity, such as the cervical muscles and the extrinsic muscles of the eyes.

Laquer (40) found that fatigue induced in one group of muscles causes fatigue in others, and Mosso has shown that this is also true of physiological fatigue. Goldflam, on the contrary, in a case which he examined, was unable to demonstrate this point. Observations on Dr. Buzzard's cases support Laquer's conclusion.

The great variations which occur in the intensity of the symptoms from time to time are very characteristic of the disease, and were first called attention to by Goldflam (13). Even in hospital, when a most careful record is kept of a patient's every act, quite unaccountable relapses occur.

Electrical reaction.—Jolly (20) in 1895 drew attention to the peculiar manner in which the muscles react to electrical currents. He found on applying a tetanising current to the

muscles of a patient suffering from this disease a brisk contraction was produced, but that if the electrodes were kept in contact with the muscle the contraction became gradually feebler until finally the muscle ceased to respond. On removing the electrodes and reapplying them again after a minute or two a good contraction was obtained, which disappeared just as on the first occasion. Jolly compared the exhaustion produced by electrical stimulation to that which results after voluntary exertion, and he gave the name *myasthenic reaction* to the phenomenon which he had observed.

The myasthenic reaction is not always present; it occurs in some cases as a passing symptom, in other cases it has not been obtained.

At one time a muscle can be exhausted in twenty-five to thirty seconds by the application of a moderately strong current, at another time it is impossible to exhaust it; a fact not surprising in view of the known variability of the myasthenia from time to time. The comparison between the myasthenic reaction and the myotonic reaction found in Thomson's disease is of interest.

Further, the myasthenic reaction is met with in different muscles at different times.

Murri (25) made the interesting observation that after a muscle has ceased to respond to the faradic current voluntary power still persists, and is only slightly diminished in degree.

The readiness with which the myasthenic reaction can be obtained, as Jolly has remarked, varies with the degree of fatigue in the muscles.

Attempts to exhaust the muscles with galvanism result only in a slight diminution in the contraction.

In cases described by Erb (2), Oppenheim (5), Bernhardt (7), Senator (10), Kojewnikov (30), Montesano (45), Wernicke (16), Pineles (23), and Kalischer (32) diminished excitability was present in certain muscles, notably in those of the tongue, neck, and shoulder. In Kojewnikov's case reaction of degeneration was present in the tongue.

Atrophy.—Although muscular atrophy is altogether ex-

ceptional in this disease, in a few cases it has been met with. Some of the recorded instances have probably been cases of polio-encephalitis (superior nuclear palsy), but others—and such cases are of extreme interest—have presented the classical features of myasthenia gravis.

In one of Erb's cases the masseters were atrophied. Atrophy of the tongue has been described by Erb (2), Dreschfeld (14), and Kojewnikov (30). In Dreschfeld's case there was some degree of atrophy in the sternomastoids, trapezii, and deltoids. In Kojewnikov's case the face muscles were manifestly atrophied, while both masseters, especially the left, were very atrophic.

In Kostetzki's (47) case the pectorals, deltoids, thenar and hypothenar eminences, glutei and spinal muscles, were atrophied. In this case, however, the diagnosis was not quite clear, the writer regarding it as a transition form between chronic bulbar paralysis and asthenic bulbar paralysis. One of Laquer's (40) cases is of the greatest importance in this relation, for in it atrophy of the deltoid, biceps, triceps, pectorals, interossei, quadriceps, and calf muscles, developed while the patient was under observation.

Fibrillary contraction was present in the tongue in cases recorded by Erb (2) and Senator (10), but in no other region has it been noticed.

In some cases occasional chronic spasmodic jerks occur in the extremities; this was so in Buzzard's female case (55).

The muscular sense and co-ordination are never impaired.

With the exception of a feeling of fatigue in the muscles after they have been exercised, and of stiffness due probably to immobility, sensory symptoms are seldom present. Sometimes patients complain, even when lying in bed, of an aching in the limbs, and this, like the stiffness, is probably connected with the immobility of the affected parts. The muscles are sometimes tender on pressure; no other objective sensory disturbance occurs.

The skin reflexes are in no way peculiar. A jaw-jerk is sometimes present. The triceps- and biceps-jerks are, as a rule, brisk, although in some cases it is difficult to elicit them.

condition of the intra-medullary portion of the anterior roots, as well as the hypoglossal root, was present, particularly well seen in Marchi preparations. Undoubtedly the condition of the nerve sheaths was pathological, but to what extent the axis-cylinders were affected could not be determined.

In the case which Kalischer describes, the diagnosis is not absolutely certain. Jolly (20) believes it to have been a case of myasthenia gravis, but Strümpell (24) is not convinced. A description of the case will be found in the appendix. Kalischer found degenerative changes in the cells of the motor gray matter from the oculo-motor nucleus, through the whole length of the cord to the sacral region, the fine fibres of the gray matter being rarified. Small hæmorrhages were seen round the dilated blood-vessels in this region. The vessels had in places undergone changes, the adventitia was thickened, and here and there aneurismal dilatation of the lumen was present.

Widal and Marinesco (33) have recorded a case in which, besides dilatation of the vessels, a partial disappearance of the chromatophilic elements in the nerve-cells of the third, sixth, seventh, and twelfth nuclei existed. Degenerated fibres were demonstrated by the Marchi method in the third, seventh, and twelfth nerve-roots. This case again is not quite typical, for death, which occurred suddenly fourteen days after the first symptoms were noticed, was preceded for eight days by slight fever.

In a case reported by Murri (25), numerous hæmorrhages were present and chromatolysis in some of the cells of the hypoglossal nucleus. Cohn disregards the cell changes found in this case, since the *post mortem* was not made until six days after death.

In two cases (Eisenlohr, 4; Wernicke, 16), recent hæmorrhages were found in the medullary nuclei. These patients died, the one of paralysis of the diaphragm, the other suddenly of asphyxia, and it is possible that the hæmorrhages were produced in the act of dying. In another case, described briefly by Charcot and Marinesco (22), in which recent hæmorrhages were met with beside

The knee-jerks are usually rather more active than normal. In a case described by Collins (26) it was found possible to abolish the knee-jerk by seven or eight strokes on the patella tendon. In one of Goldflam's cases a similar condition was present, while in Strümpell's case the activity of the knee-jerk appeared to diminish somewhat. In other cases the knee-jerk did not diminish after repeated tapping (Laquer, 40; Angelini, 49; Buzzard, 55). The knee-jerk does not diminish if the quadriceps is fatigued. In one of Buzzard's cases exhaustion of the vastus internus by faradisation produced no effect on the knee-jerk. Ankle clonus is not met with. The plantar reflex, where it has been tried, gives a flexor response.

The sphincters are never affected in this disease. No trophic changes occur in the bones or skin.

Morbid Anatomy.

In the great majority of cases in which an autopsy has been made no lesion has been found.

Of seventeen cases which have come to the *post-mortem* table, in only six was anything abnormal found which could *possibly* account for the symptoms, notwithstanding that in many of the cases the most modern methods were used, and the microscopical examinations made by highly skilled observers.

Two cases described by Mayer (18) and Kalischer (32) respectively, deserve special attention in this relation. Mayer's case appears to have been an undoubted example of the disease. The patient had speech trouble, difficulty in swallowing, weakness of the jaw muscles, double ptosis, and paresis of the extremities; there was no atrophy, and the reflexes were normal; towards evening and after exertion all the symptoms were most pronounced; temporary improvements and relapses occurred, death ultimately occurring by choking. Microscopical examination showed that the cells of the hypoglossal nucleus, with the exception of a small number which contained vacuoles, were completely normal, as were those of the anterior horns. A morbid

in some cases is opposed to the view that the disease is always a purely muscular affection. Strümpell (24) argues from the myasthenic reaction that the affection is not limited to the upper motor neuron.

It is not possible, with the facts we at present possess, to make any definite statement as to the part or parts of the neuromotor apparatus involved by the morbid process.

Pathology.

We are equally ignorant as to the true pathology of the disease. It has been suggested, and this view is very generally accepted, that the disease is due to some toxic agent, the result of a disturbed metabolism. Laquer strongly supports this metabolic theory, and he points out with regard to physiological fatigue that the preservation of normal muscle excitability is dependent upon a suitable change between rest and activity, that long-continued activity, resulting in the complete loss of irritability, is the condition in fact which we call fatigue, and that the appearance of fatigue depends upon the existence of products of decomposition out of the muscle substance itself which accumulate in the blood.

Collins (26) thinks that the most plausible theory of the pathogenesis of the disease is that a poison is generated in the system and continuously operates on the vegetative portion of the body. When vitality is sufficiently low to let the poison get the upper hand a relapse occurs.

It may be, however, that the toxine is of exogenous and not of endogenous origin. As Strümpell points out (24), the absence of any family tendency and the onset in some cases after an acute illness are in favour of this view.

In regard to the pathology of the disease it is impossible at present to do more than surmise. The view we ourselves put forward is that the disease is due to a poison probably of microbic origin acting upon the lower motor neurons and interfering with their functional activity without necessarily producing discoverable change in structure. We suggest that the poison acts upon the motor fibre (= axon) or end

the aqueduct of Sylvius and about the ventral nucleus of the vagus, the patient died with "bulbar symptoms."

Such cases are, however, exceptional, and, as already observed, in the majority of cases no visible lesion has been met with after death. In the few cases in which changes have been present, these changes, as Kalischer (32) and Laquer (40) have pointed out, were not present in all those centres whose functions showed signs of disturbance during life. Changes, no doubt, are present, although it may be that they cannot be demonstrated by our present methods; but it is open to question whether those hitherto described are actually the cause of the symptoms which the patients manifest during life. It is of great importance that in future careful examination be made not only of the multipolar cells, but also of the muscles themselves, of the nerve-endings in the muscles, of the motor nerves, nerve-roots and cortical cells.

In the few cases in which the muscles have been examined no changes have been found.

Seat of Lesion.

It can be readily understood that in the presence of such a meagre morbid anatomy, various views are held as to the seat of the lesion.

As Strümpell states (24), there is not a single case which is associated with symptoms pointing to parts other than the motor nervous system, and so much seems to be admitted by all observers. Collins (26), although he admits that the phenomena of the disease are largely manifested through the motor system, is inclined to regard the condition as due to an affection of the sympathetic. He bases his opinion upon the attacks of collapse and cyanosis from which his patient suffered.

Jolly (58) concludes from the myasthenic reaction that an abnormal condition exists in the muscles themselves. There are, he contends (58), no certain facts proving that the disease is one of the central nervous system.

Mendel (58) thinks that the occurrence of sudden death

plates; as to whether it also acts upon the cell-body (in bulbar nuclei or anterior horns), we can form no opinion.

The evidence in favour of a toxine being the pathogenic agent is strong. The symmetry of the affection is alone highly suggestive of this view; also the fact that it occurs in connection with disease in which nerve poisons are known to be produced. Thus cases occur after such diseases as scarlatina, influenza, sore-throats, typhoid fever, which are well known to be often followed by toxic neuritis. Other cases have occurred after otitis media, others have begun with diarrhœa, both of which diseases may be associated with blood-poisoning. We have ourselves seen Laundry's paralysis originate in an acute gastro-intestinal attack with profuse diarrhœa, undoubtedly due to ptomaine poisoning, the poison in this instance apparently paralysing, like urari, the motor end organs. Megrim, again, is a disease which is almost certainly toxically induced, and it is noteworthy that one of the cases of myasthenia gravis occurred in a megrimous patient immediately after an attack of megrim.

It is now fairly well established that tabes dorsalis is a toxic disease, the poison acting chiefly upon the sensory periphery, but also to some extent upon the lower motor neurons, causing the Argyll-Robertson phenomenon, paresis of the extrinsic ocular muscles, and the levators palpebrarum. Now, in the case of the latter muscles, the paresis is often transient; moreover, we have observed the fatigue phenomenon in these cases. Seeing then that transient paralysis and the fatigue phenomenon may be brought about by a toxin, it does not seem improbable that myasthenia gravis may be similarly induced. Assuming the disease to be produced by a toxin, we have to inquire whether the poison is endogenous or exogenous. We know that substances normally present in the body may cause fatigue, and conceivably myasthenia may be due to an excess of such poisons —poisons, namely, produced by the metabolism of the organism. The occurrence of the disease after such maladies as influenza, scarlatina, typhoid, and sore-throat suggests a microbic origin.

The poison may conceivably act upon the motor nervous

system, or upon the muscles themselves. In the former case it might involve the upper or the lower neuron or both.

Several arguments may be adduced against the upper neuron being the essential seat of the lesion. The myasthenic reaction and the exhaustibility of the knee-jerks, as met with in Collins' case, the occasional atrophy and diminished faradic excitability, cannot be explained on the assumption of an upper neuron-lesion alone. Nor can we in this way explain the tendency to spontaneous attacks of dyspnœa, attacks so severe as to frequently prove fatal. Nor, again, the tendency to choking from pharyngeal paralysis, seeing that the choking is essentially due to defect of the voluntary part of the act of deglutition. Further, the attacks of palpitation are in no way suggestive of a lesion in the upper motor level. One cannot, indeed, fail to detect a likeness between the attacks of dyspnœa, choking, and palpitation met with in myasthenia gravis, and similar attacks occurring in affections of the lower motor level, to wit, bulbar paralysis and peripheral neuritis—the diphtheritic form, for instance.

But perhaps the most conclusive evidence against the view that the essential lesion is situated in the upper motor neuron is afforded by the myasthenic reaction ; the muscles in myasthenia gravis become unduly exhausted by faradic stimulation, the same fatigue being experienced as occurs after voluntary action in this disease. It is evident that this phenomenon must be due to a peripheral cause.

We have next to consider whether the disease involves the lower motor neurons or the muscles. On the assumption that it is of toxic origin, it seems safe to exclude the muscles from an essential share in the pathological process. We have no facts in pathology suggesting that a poison can exist in the blood in sufficient quantity to seriously impair the functional activity of the entire muscle system, and this without producing any other symptoms, much less that such a poison can pick out symmetrical muscles, *e.g.*, the levators palpebrarum, to the exclusion of all others. What we know of the action of drugs and toxins strongly suggests that if

myasthenia gravis is due to a *poison*, acting selectively, it produces its effects by acting upon nerve rather than upon muscle.

The myasthenic reaction, while it does not enable us with certainty to fix the lesion upon nerve rather than upon muscle, yet lends support to the view that the former is involved. On this assumption the motor-fibre, end-plate, or muscle-fibre may be implicated. The characteristic reaction is obtained by faradism, not galvanism. Now faradism causes contraction essentially through the nerves; its power of causing contraction by direct stimulation of the muscles is small. The inability of galvanism to cause the exhaustion does not help us, since it contracts the muscles both through the nervous system and by direct muscle stimulation; this inability is doubtless due to the fact that it cannot induce tetanus. The crucial test would be to find if, after inducing complete myasthenia by faradism, the myasthenic muscles respond to galvanism. Such response would afford fairly conclusive evidence that the exhaustion was one of the motor nerve-fibres or end organs.[1]

We have then arrived thus far: the motor end-fibres or end-plates are especially involved in myasthenia gravis. Is the body of the neuron also involved? We can conceive of the toxine acting upon the whole of the lower motor neuron, cell-body, axon, and end-plate. It is indeed possible that it may act primarily upon the cell-body, the axon being secondarily affected through withdrawal of the normal trophic influence emanating from the cell-body. The muscle-atrophy and diminished faradic excitability which sometimes occur prove that the lower motor neuron is, at least occasionally, involved.

[1] We have, at present, no marked case of myasthenia gravis under observation upon which to make this observation. On the suggestion of one of us (H. C.), Dr. Farquhar Buzzard kindly undertook the following experiment:—In a well-marked case of myasthenia gravis a moderate galvanic current was applied to the biceps muscle and a contraction obtained. The muscle was then faradised until it gave no response to a strong stimulus. Then it was tired out by making the patient flex the elbow against resistance, exerted until all power of flexion was lost. On applying the same strength of galvanic current as used at first an excellent contraction was obtained. Finally, on again applying the faradic current, the muscle was found to be still irresponsive.

The fact that discoverable lesions are generally absent *post mortem* is just what we should expect from the symptoms of the disease. Were a permanent organic lesion present we should not expect such marked variations in the degree of paresis to occur as characterise the disease—complete ability to move the muscles one moment, absolute paralysis of them a few moments later, or an entire disappearance of the symptoms for weeks, months, or years together.

What we know of the action of drugs entirely supports the view that function may be profoundly modified by the action of chemical substances, without any discoverable organic change being wrought. Nothing is more certain than that antagonistic poisons may set up a countless variety of transient perturbations in the nervous system. Witness, *e.g.*, the nervous phenomena observed in dyspepsia, and among these it may be observed that lassitude and muscular fatigue are not infrequent.

But while numerous nervous symptoms may result from blood-poisoning independently of discernible organic lesion, it is a fact that in all cases hitherto known to us of paralysis thus resulting, a well-marked lesion is discovered *post mortem*; we allude to such diseases as peripheral neuritis and poliomyelitis. And herein lies the pathological interest of myasthenia gravis. In it we have, we would suggest, an instance of a toxic paralysis, lasting intermittently for a long period of time, and often terminating in death, and in which, nevertheless, no discoverable lesion is necessarily induced. The wonder is not that such cases are rare, but that they are not more frequent. Purely functional nervous symptoms of toxic origin, such as numbness, tingling, twitching, drowsiness, coma, are common enough; why should not purely functional toxic paresis be equally common? As a matter of fact, we believe that such pareses are not altogether uncommon in a very mild form, more especially in connection with dyspeptic toxæmia.

What we suggest, then, is this—that in myasthenia gravis, a toxin, probably of microbic origin, circulates in

the blood, and acts selectively upon the lower motor neuron, so as to modify its functional activity. This change in functional activity is such that the lower neuron soon becomes exhausted by the transmission down it of stimuli, whether originating in an act of will or started by the faradic current. As to which part of the neuron the poison acts primarily upon one can only vaguely surmise; but we lean to the view that it is upon the axon. This brings the disease into line with such a disorder as diphtheritic neuritis. Imagine an attenuated toxin, such as causes this latter disorder, to remain for some time in the body, and the effect might be much the same as is observed in myasthenia gravis. In the earlier stages of the disease no organic changes would be produced, but after prolonged action one might expect such changes to occur, and be accompanied by atrophy of the muscles and diminished faradic excitability.

Diagnosis.

The diagnosis in well-marked cases of myasthenia is easy. The paresis and speedy fatigue of voluntary muscles on exertion, the myasthenic reaction, the absence of muscular atrophy and of fibrillary tremors, of objective sensory disturbance, trophic changes and sphincter affection, together with the remarkable variations in the intensity of the symptoms—form a distinct clinical picture.

A diagnosis can often be made at first glance from the facial expression, the ptosis, and nasal speech.

The diseases most liable to be confounded with myasthenia gravis are hysteria, bulbar palsy, polioencephalitis, the muscular distrophies, amyotrophic lateral sclerosis and general asthenia.

No doubt in hysteria and neurasthenia ready fatigue after exertion is met with, while the variations in the symptoms, the presence of ptosis, difficulty in swallowing, and attacks of breathlessness, all occur. In such cases absence of a nervous heredity and hysterical manifestations, the absence of sensory symptoms and the presence of the

myasthenic reaction, should enable a diagnosis to be arrived at.

Cases undoubtedly occur in which the diagnosis is very difficult.

A case recently seen by one of us illustrates the difficulty which may arise in diagnosing early cases. The patient was a young married woman, who for three years (since her last confinement) had suffered from general muscular weakness. When first seen, after ascending a long flight of stairs, she was much exhausted and very breathless, speech was somewhat nasal in character, and she had slight double ptosis. She complained of general exhaustion after any slight exertion, had had definite diplopia on several occasions, and said she had been very anæmic ever since her confinement. No weakness in the jaws or trouble with deglutition existed, and although the symptoms were very suggestive the facts did not justify a certain diagnosis, especially in the presence of a marked anæmia.

In the myopathies fatigue after exertion is often complained of. We have already seen that the facial appearance is very similar to that seen in the Landouzy-Dejerine form of myopathy. The waddling gait and the tendency for the paresis to affect the proximal muscles of the extremities are other points of similarity; but, as Oppenheim (58) remarks, the resemblance is superficial only, for it is only in myopathic patients, who show the fatigue phenomenon, and those in whom the muscles are only slightly wasted, that any confusion is likely to arise (Jolly, 58). Toby Cohn (58) argues from Laquer's case, in which muscular atrophy developed, that the relation to the myopathies is closer than would appear at first sight.

Wernicke's case (16) was diagnosed at first as one of amyotrophic lateral sclerosis with bulbar symptoms, the absence of muscular atrophy being specially remarked upon.

From bulbar palsy the disease distinguishes itself by the presence of ptosis, upper facial weakness, paresis of the jaw and neck muscles and of the extremities, and by the myasthenic reaction. The variation in intensity of the symptoms is a diagnostic point of great importance.

Lastly, the differentiation from polioencephalitis has to be considered. Here the diagnosis may be extremely difficult. The mode of onset, absence of muscular wasting, and above all the variability of the symptoms, are points to which especial attention must be directed.

Prognosis.

Of sixty recorded cases twenty-three ended fatally.

The average duration of life of the fatal cases was about a year and a half after the development of the first symptoms.

These figures probably give an exaggerated impression as to the fatality of the disease.

In one case, that of Widal and Marinesco (33), death occurred fourteen days after the first symptoms were manifested. In Dreschfeld's case (14) the patient lived fifteen years.

That cases improve in a remarkable way and remain free from symptoms for months, or even years, is certain. That complete recovery sometimes takes place is probable. Goldflam (9) described his cases as belonging to "a curable form of bulbar paralysis." In one of Erb's cases (2) the patient was in good health four years after his symptoms had disappeared. In some cases the disease lasts a number of years.

Involvement of the respiratory muscles, with consequent attacks of dyspnœa, is a symptom of the gravest significance, and the patient's friends should be warned that death may occur suddenly during one of these attacks.

Any pulmonary disorder, whether chronic or acute, is to be considered as of very serious import.

Treatment.

No specific treatment is known for the disease. The patient must be enjoined to take as much rest as possible, and in severe cases should be confined to bed.

Excitement of every sort is to be avoided, inasmuch as the symptoms are much increased by it.

Since cold appears, in some cases at least, to aggravate the disease, attention should be paid to the patient's clothing. Cold baths are to be avoided. The patient's whole life is to be regulated so as to avoid muscular fatigue.

The diet should be nutritious, and if there is any difficulty either in deglutition or mastication, the food is to be carefully chopped, to obviate the necessity for chewing, and to avoid, as far as possible, the risk of choking.

In the event of the necessity arising for using the stomach tube, the greatest care must be taken, for, as in other forms of bulbar palsy, the procedure is not unattended with risk; indeed, in a case reported by Oppenheim, a fatal result followed the passage of the tube.

As to drugs, little can be said. Erb and Goldflam's cases were treated with iodide of potassium and mercury, but there is no evidence to show that this treatment actually has a beneficial effect.

Strychnine was employed by Strümpell (24) in large doses without result. In Buzzard's cases hypodermic injections of strychnine up to one-sixth of a grain in the twenty-four hours were given without benefit.

Faradism, galvanism, and massage were all given an extended trial in Buzzard's male case. Faradism appeared to do harm, the patient feeling weaker as the result of the treatment, while galvanism and massage did no good. Galvanism to the spine has been recommended by Erb (2) and Goldflam (13), but appeared to have no positive value in the case already referred to.

Suprarenal extract was also ineffectual. Under thyroid tabloids, together with a daily injection of five minims of solution of strychnine, the patient improved so much that after six weeks he felt almost well. The improvement began on the day the thyroid was commenced, but it is quite possible that suggestion may have accounted for the success which attended this line of treatment; the case relapsed while still taking the tabloids.

In another case thyroid extract was given without benefit.

Iron and arsenic appear to be of use as general tonics, although they have no specific action upon the disease.

NOTES OF SIXTY REPORTED CASES OF MYASTHENIA GRAVIS.

Case 1, Wilks (1).—A stout girl, looking well, came to the hospital on account of general weakness; she could scarcely walk or move about, she spoke slowly and had slight strabismus. She remained in this state about a month, being neither better nor worse; every movement of her limbs and her speech were performed so slowly and deliberately that the case seemed rather one of lethargy from want of will than an actual paralysis.

At the end of this period the symptoms became aggravated, and in about three days they had assumed all the well-marked characters of bulbar paralysis. She spoke most indistinctly, swallowed with great difficulty, and was quite unable to cough. The limbs were, however, not paralysed, she was able to get out of bed. It was shortly afterwards seen that her respiration was becoming affected, the difficulty of which rapidly increased and in a few hours she died.

The medulla oblongata was very carefully examined and no disease was found. It appeared quite healthy to the naked eye, and the microscope discovered nothing abnormal.

Case 2, Erb (2).—Man, aged 55. Illness developed slowly with pains in the nape of the neck; after four months he could not hold his head up; the muscles of mastication were weak, and he had double ptosis. Thereupon ensued weakness in the tongue and difficulty in swallowing; the mouth was kept open. There was atrophy of the neck muscles and trapezii, and slight diminution in the response to faradism in the trapezii, masseters and splenii. There was a high degree of auditory hyperæsthesia on the left side. Decided improvement occurred after a six months' course of galvanism. He could hold his head up, swallow well, and masticate much better. After a few months he became well, though he still had occasional weakness in the arms.

Case 3, Erb (2).—A peasant woman, aged 30, was seen on October 30, 1870. For nine weeks she had had slight twitchings in the face, diplopia and ptosis; later, difficulty in mastication, stiffness in jaws, much headache and giddiness developed; recently, she has suffered from palpitation and weakness in the limbs. She has had blinking of the eyes and twitchings about the mouth for a month.

State.—The eyes are half closed, the movements of the eyeballs are very defective, there is no diplopia, she has difficulty in

opening and closing the mouth, the mouth is constantly open, and there is some stiffness and weakness of the facial muscles. A feeling of weakness exists without actual loss of power in the lower extremities. She has some left auditory hyperæsthesia.

November 4.—The symptoms are worse. She complains of difficulty in swallowing, and of a profuse salivary flow.

November 10.—There is improvement in the eye muscles and muscles of mastication. She says that for many weeks the head has felt heavy and she has had difficulty in holding it up, while there has been a feeling of weakness in the posterior cervical muscles.

December 22.—Great improvement.

January 17, 1891.—She is slightly worse. The tongue appears atrophied. During the next month fluctuations in the severity of the symptoms occurred.

July, 1891.—Is much worse again. She now has ptosis, diplopia, difficulty in mastication, and atrophy of the masseters. The movements of the soft palate are sluggish; speech is nasal; utterance difficult; the voice is weak, especially after lengthy speech. There are great weakness and fatigue in the legs; she has frequent palpitation. Pulse 100.

Improvement again occurred, but later she became worse and died suddenly on the night of April 24, the illness having lasted eight months. There was no *post mortem*.

Case 4, Erb (2).—A merchant, aged 47, was seen on July 11, 1871. During the last year he had suffered from pain in the occiput, and stiffness and weakness in nape of neck. Since April last he has had sharp pains in head and nape of neck, vertigo and occasional dimness of vision. Towards the end of May weakness in the arms and legs compelled him to remain in bed; he could with difficulty hold his head up. In June he developed double ptosis and was very sleepless; during the last few weeks has been better.

Present condition.—A powerful man. He has slight paralysis of the upper face, he cannot wrinkle the forehead well and there is slight twitching of frontalis. The head is bent forward. The right side of the palate is weak; swallowing is unaffected, but there is occasional fatigue of masticatory muscles. Walking soon causes fatigue, while vertigo and dimness of vision occur after walking much.

No improvement followed fourteen applications of galvanism.

Case 5, Erb (2).—A male, aged 48. His previous health was good. Six months ago the lips felt stiff, and he had

headache and diplopia. Three weeks later he had pain in the neck and the head fell forward, later there was difficulty in chewing and swallowing. Pain and weakness in arms and legs, giddiness and ringing in the ears developed. The voice became hoarse and the movements of the tongue difficult, while ptosis developed.

Present condition.—He has difficulty in speaking, chewing and swallowing. The pupils react normally, the extrinsic eye muscles are unaffected. There is weakness in the right arm, and fatigue is easily produced. He has severe pains in both arms and legs at times. Fibrillary twitchings are seen in the tongue, which is slightly atrophied.

The patient went out of the Klinik much improved after fifty-two applications of the galvanic current.

Case 6, Eisenlohr (4).—A girl, aged 18. *As a child she had suffered from megrim.* This was *occasionally accompanied by ptosis. Two years ago she developed sudden diplopia,* which disappeared after a three months' course of iodide, but returned in the summer of 1885; sometimes chiefly on one side, sometimes on the other. In July, 1886, after an attack of megrim, she had weakness of both hands. In August there developed slight weakness of the legs, breathlessness on quick movement, and difficulty in swallowing, chiefly at midday and in the evening, also weakness of cervical and masticatory muscles. All these symptoms varied in severity even in the space of a single day.

August 16.—The patient was examined. She has ptosis, chiefly left-sided, with little movement of the eyes; can only close the eyes feebly; there is double facial weakness and little movement of the soft palate; she can only swallow small quantities of fluids, which readily regurgitate through the nose; she has great difficulty in moving the head; the respiratory movements are defective and the cough is weak; there is paralysis of all four extremities; swallowing is easy in the morning, difficult in the evening, and movements of tongue are fatiguing. Respiration is superficial; pulse frequent.

August 18.—Palpitation; pulse 140; frequent respirations with fruitless efforts to expectorate accumulation of mucus, &c., from the throat.

August 21.—The diaphragm is almost entirely paralysed. Death.

Post mortem.—The cortex cerebri was not examined. Hyperæmia with small recent *ante-mortem* extravasations was found in the medulla, numerous small but not degenerated fibres

(fasern) in the roots of the hypoglossal vagus and accessorius were observed.

Case 7, Oppenheim (5).—A maidservant, aged 29, seen February, 1885. *Nine months ago she noticed weakness in the hands*, which gradually increased and involved the legs also. For the last four weeks she has noticed fatigue during speaking and has often to pause, also weakness of lips and difficulty in swallowing.

Present condition.—There is little movement of the facial muscles during speech; voluntary movement of the lower face is very defective; there is weakness in opening and closing the jaws; mastication rapidly causes fatigue; she cannot move the lower jaw laterally; there is defective movement of the soft palate during phonation; there is tremor of the tongue with defective movement; speech is nasal. She cannot cry out; inspiratory movements are defective; she complains of breathlessness; there is great weakness in arms and in the abdominal muscles; she says that the degree of weakness in the extremities varies greatly from time to time. Sight is normal. There is no muscular atrophy, no electrical change, but the deltoid and biceps require unusually strong currents to excite contraction.

June, 1885.—Manifest dyspnœa. In the following month attacks of difficulty in swallowing and speaking developed.

August, 1885.—She has weakness in the orbicularis palpebrarum, and attacks of dyspnœa; the paralysis is increasing with slight remissions.

January, 1886.—She complains of pains in nape of neck, occasionally in the extremities and face.

May, 1886.—There is increased weakness in extremities, she can scarcely hold her head up.

September, 1886.—She died in a dyspnœic attack with pneumonia.

Case 8, Lauriston E. Shaw (6).—A baker, aged 37. He had enjoyed good health until the present illness. Six months ago he had a severe attack of diarrhœa lasting a fortnight. Ever since then he had had a feeling of weakness, especially in the muscles of mastication, the arms, and to a less extent, in the legs. After this difficulty in articulation and swallowing supervened. The weakness was slight in the morning and increased as the day advanced. He has had two attacks of dyspnœa within the last fortnight; there has been a copious secretion of frothy mucus with difficulty of expectoration.

Admitted, February 12, 1887.—He can barely stand; he says

he often falls down. The soft palate is normal. The mouth is constantly held slightly open. There is loss of power in the lower facial muscles. The naso-labial fold is obliterated, causing a vacant expression. There is some difficulty in protruding tongue; the masseters are weak; he cannot hold a pipe either with the lips or teeth; has difficulty in swallowing and occasional diplopia; the tendon reflexes are brisk; spurious ankle clonus is present on the right side; the cremasteric and plantar reflexes are normal, the abdominal and epigastric absent. The mental condition is good; he has perfect control over the bladder and rectum.

He died shortly after admission from dyspnœa, apparently resulting from paralysis of the muscles of respiration.

Post mortem.—Pons, medulla, and cord were examined by Dr. Newton Pitt, and nothing abnormal discovered.

Case 9, Bernhardt (7).—A joiner, aged 44. In August, 1886, he first complained of fatigue and weakness in the eyes, later he had diplopia.

Present condition.—November, 1886. There is double ptosis, on the right side chiefly; the left external rectus is paralysed. After chewing for a short time he cannot close jaws. Under potassium iodide and galvanism he was dismissed cured in February, 1887, and continued well till 1890.

July, 1890.—The head falls forward on chest, chewing and swallowing are difficult, and speech soon becomes nasal; the lips are fatigued after continued speech, and work easily exhausts him. Movements of the arms are difficult; there is slight right ptosis; the electrical reactions are normal, but there is slight quantitative diminution in the neck muscles.

September, 1890.—He has dyspnœa on walking quickly, or on mounting steps. *The right vocal cord moves less readily than the left.* He improved under potassium iodide and electrical treatment.

1890.—Sudden death occurred. No *post mortem* was allowed.

Case 10, Jolly (8).—A tanner's apprentice, aged 15, was admitted on August 19, 1890. The family history was unimportant. He had suffered from middle ear disease for a few years, and had had no other illness of importance. His present trouble began eight months ago with weakness in the legs; later, weakness in the arms, difficulty in speaking, giddiness, and pain in the neck and sacrum developed. After walking a short distance he requires support to prevent him falling. He has double

ptosis; fatigue soon develops after whistling, speaking, chewing, or swallowing; the eye movements are good. The knee-jerks are somewhat increased. There are no qualitative electrical changes, but the quantitative estimation is peculiar, the muscles at first respond well, but if the same muscle is stimulated repeatedly so that a tetanic contraction is produced, the contraction of the muscle gradually becomes weaker until it is only elicited by very strong currents—in other words, the reaction of fatigue is present.

The patient died suddenly on March 18 while eating, a piece of food sticking in his throat. At the autopsy nothing abnormal was found either in the nervous system or muscles.

Case 11, Jolly (8).—A boy, aged 14, who had been strong and healthy until the summer of 1893. At that time (one and a half years ago), while at school he noticed that he could not see the black-board properly, for he was unable to keep his eyes open, the eyelids drooping if he looked at an object for any length of time. After a rest, and particularly in the morning, this was not the case. Later, tiredness of the legs, also of the arms, of the neck and lip muscles, appeared. While eating he had to stop frequently because he could not go on chewing. If he read aloud, speech became indistinct, reminding one of general paralysis; there was no affection of the tongue or of swallowing. All voluntary muscle was readily exhausted, and also the exhaustion of one group of muscles was seen to produce fatigue in others; if, for example, the legs are tired, then the arms become fatigued, though not to the same degree.

Case 12, Senator (10). (*Doubtful case.*)—A workman, aged 41. Twenty years previously he had an ulcer on the penis and swelling of glands; no other evidence pointing to syphilis. Been feverish for nine weeks and had a slight cough, rigors, heats, sweats, and nasal speech. On the night of November 27 and 28 he had difficulty in swallowing and speaking, tongue was immobile, and there was little power of moving the jaws, a fixed expression, and the eyes seemed large. November 29 he could not move his right arm, there was a tendency to walk to the left and he drags the right leg.

Present condition.—He cannot speak; pulse 64; respiration 28. He can only move the right arm slightly; the right leg is paralytic; the plantar and patellar reflexes are active; rather more on the right side. There is paralysis of all the muscles supplied by the facial nuclei; laughing and whistling are im-

possible. He cannot close his eyes properly. He keeps the mouth half open and cannot powerfully close it. The lateral movements of the jaw are unaffected; the tongue is motionless; he has great difficulty in swallowing; the electrical reactions are normal, but the left facial muscles require a stronger current than the right. Involuntary twitchings of the mouth occur; there is diminished movement of the soft palate; the eyeballs are prominent.

The condition gradually improved with slight fluctuations till February, 1885, when he got worse. Death occurred from exhaustion in March, four months after beginning.

Post mortem.—A microscopical examination of the base of the brain, cord, peripheric nerves and muscles, revealed nothing morbid.

Case 13, Senator (10). (*Doubtful case.*)—A woman, aged 36. She is very anæmic and is suffering from sarcomata of the ribs. She developed in the course of the illness a nervous affection, pains in the head, back, and lower extremities, together with a feeling of fatigue. At this time albumosuria was first detected. She complained of difficulty in swallowing; she said that her chin felt as if it were dead and "did not belong to her." It was found to be quite anæsthetic. The tongue was almost completely paralysed; it was not atrophied, though fibrillary twitchings often seemed to occur about the middle of the tongue. The pulse rate was increased. The electrical reactions in the face and lower limbs were normal; in the tongue excitability was diminished. Death occurred somewhat suddenly.

At the *post mortem* multiple sarcomata of the ribs, double fibrinous pleurisy and chronic parenchymatous kidneys with waxy change; neither in the brain nor medulla was anything found to account for the condition.

The severe anæmia, Senator thinks, may have produced the symptoms by acting upon an imperfectly developed nervous system. Senator remarks that nervous symptoms have been present in several cases of multiple myeloma, *e.g.*, in Stockvis's case there was defective speech and swallowing, together with salivation, paralysis of the facial and trigeminus nerves, and paraplegia.

Case 14, Hoppe (11).—A blacksmith, aged 40, family history good, no syphilis. The illness began by double ptosis, followed by weakness of the muscles of mastication, and difficulty of speech and swallowing. Examination showed paralysis of the

right internal rectus and left superior rectus, the upper and lower part of the facial; great weakness of the masticatory muscles; little movement of the soft palate, paralysis of adductors and abductors of vocal cords; great weakness of the extremities.

The electrical reactions were normal, and there was no muscle atrophy.

The patient died, and no pathological lesion was found *post mortem*.

Case 15, Remak (12).—A girl, aged 12. She had influenza in February, 1890, with suppuration of the left ear. In July it was noticed that the eyes did not close during sleep, and speech was often indistinct. In September she complained of difficulty of swallowing. She had a fixed facial expression, the tongue was weak and the soft palate sluggish. Speech was nasal and indistinct, and after speaking a little while she could not articulate at all because of weakness in the tongue and lips. The difficulty in speech and swallowing improved under galvanic treatment. She had difficulty in getting up expectoration, and often dyspnœa.

October 6.—There is slight tremor of tongue, she cannot raise the tip.

October 16.—She has slight weakness of the arms.

October 21.—Pulse 120. Severe dyspnœa in consequence of weakness of the respiratory muscles; swallowing is impossible.

November 5.—Intense dyspnœa; death. No *post mortem*.

Case 16, Goldflam (13).—A male, aged 25, house-servant, was seen on December 22, 1891. His illness began suddenly seven weeks ago, with headache, giddiness and limitation of the movements of the head. A week later disturbance of speech and swallowing set in, followed by weakness in the arms—a few days later in the legs. Recently he has had severe pains in the shoulder and sacrum. There is no history of syphilis or alcohol.

Present state.—A fairly well-developed man; his speech is nasal. There is very little mobility of the soft palate; the pharyngeal and laryngeal reflexes are markedly diminished; swallowing is difficult; fluids frequently regurgitate through the nose; mastication is fatiguing. The lateral movements of the lower jaw are feeble, he cannot open the mouth widely; the tongue movements are good; lagophthalmos is present, greater on the right side; the conjunctival reflexes are diminished; the pupils react well; the neck muscles, sterno-mastoids, and trapezii are weak.

The patient cannot lift his hands up to the horizontal. The respiratory excursions are feeble. He does not complain of dyspnœa. The legs are feeble. The deep reflexes increased. There is no ankle clonus and no atrophy or fibrillary twitchings, while the electrical excitability of the muscles is normal. There is no affection of bladder, rectum or sensation.

January 9, 1892.—The abdominal muscles are feeble. The knee-jerks are poor and more difficult to elicit the longer one tries.

January 25.—The patient feels weakest after sleep, after a few movements he is better. In blowing the nose air comes through the mouth.

February 8.—The knee-jerks become feebler the longer the tendon is struck, that on the right side finally disappearing.

February 10.—It is especially the large muscles near the trunk which are affected. Tiring of the extremities diminishes the power in the others.

January 4, 1893.—He remains now practically well.

Treatment.—Galvanic stream to the spinal cord. Potassium iodide, iron and arsenic internally.

Case 17, Goldflam (13).—A male, aged 25, was seen in April, 1892. Ten years ago he had typhoid, which lasted three months. A mental affection followed. He refused to take food, was morbidly shy and tried to commit suicide. The present illness began four months ago, without observable cause, with weakness in the arms and legs. These symptoms developed in the course of two weeks. Shortly after he had difficulty in chewing. The neck muscles became affected, and giddiness developed. A month ago he had a very bad attack of dyspnœa and general loss of power of movement.

State.—A well-developed man. There is paresis of the lower face; he cannot whistle. He can shut the eyes well; the tongue and palate act normally. Chewing is difficult. There are no psychical disturbances; patient is, however, anxious about his condition. The neck muscles are feeble; the deltoid and upper arm muscles are most affected, while in the lower extremities the flexors and extensors of the hip are chiefly involved. The tiring of one extremity appears to have no influence upon the others. The deep reflexes are increased; there is slight ankle-clonus. The muscles all react normally to electricity. The symptoms vary from day to day.

July 1.—The deep reflexes are somewhat difficult to obtain.

March 16, 1893.—The patient is now almost well. He has had two relapses, both brought on by a fright.

May, 1894.—The patient only remained well for six months; the symptoms are again present.

Case 18, Goldflam (13).—Governess, aged 22, was seen on April 13, 1892. With the exception of anæmia and headache, especially during the last winter, she has always been healthy. The present trouble began three weeks ago without discoverable cause, with headache, photophobia, trembling in the eyelids, and diplopia. A week later she had a feeling of oppression, then of weakness and heaviness in arms and legs; later still, right-sided ptosis and difficulty in chewing, the jaw tended to fall down and had to be supported by the hand. Fluids came through the nose. The voice became nasal. There were no signs of hysteria. The patient's father died of a brain tumour; her mother had a congenital divergent strabismus.

State.—A fairly well-developed, rather pale girl; internal organs normal; pulse 90. Except for the right-sided ptosis, the eyes are normal. There is lower facial weakness and paresis of the palate, its reflex excitability being diminished. The tongue is somewhat thin, but its mobility is normal. Speech nasal, and chewing difficult. The movements of the head are weak. The head tends to fall forward. The deltoid appears to be the weakest muscle of the arms. There is very little weakness in the lower extremities. The electrical excitability of the muscles is normal. The deep reflexes are very active. She suffers from attacks of dyspnœa.

May 2.—Swallowing is difficult.

September 20.—She is much better; the nasal voice is still present, but no other symptoms of the disease.

Case 19, Dreschfeld (14).—A widow, aged 36. Fifteen years ago she developed sudden double ptosis, but completely recovered in six months. Ten years ago she had a second attack of ptosis with diplopia, followed by difficulty in speaking and swallowing, and weakness in the arms; she had to stop eating frequently because her jaws ached; she could not properly open her mouth to protrude the tongue, and she lost the power of whistling. The eye symptoms improved, but about nine months afterwards she suddenly became worse and was admitted.

On admission.—The intrinsic ocular muscles are normal; she has ptosis, and the extrinsic muscles supplied by third nerves are nearly completely paralysed; while the fourth and sixth on both sides are completely paralysed; the masseters, pterygoids and temporals are much weakened; she cannot close the eyes,

wrinkle the forehead, or frown, press the lips together, smile or whistle.

The movements of the tongue are very limited, it is somewhat atrophied; the depressors of the hyoid are weak; the soft palate is paralysed, there is no palatal reflex; there is difficulty of swallowing; great difficulty in clearing fauces and posterior nares of saliva and mucus. Difficulty in articulation; the laryngeal abductors are weak; the sterno-mastoids and trapezii are somewhat atrophied; the diaphragm acts slightly; the deltoids are atrophied and paralysed; there are no fibrillary twitchings; pulse 130; the wrist and triceps reflexes are markedly increased. The facial muscles show no reaction of degeneration.

November 22.—The patient is much worse; she is markedly cyanosed, the respirations are spasmodic and dyspnœa is great; swallowing is impossible, and there is no longer true articulation; the patient can only produce a faint grunt. The pulse is at times intermittent, weak and thread-like. During the night the breathing became much more laboured, the temperature rose to 99·5, and the patient gradually sank and died.

Post mortem.—Careful microscopic examination revealed nothing. The muscles were not microscopically examined.

Case 20, Suckling (15).—A barmaid, aged 21, complained of great general weakness. A brother has epilepsy, a sister paralysis. The patient's illness commenced three years ago with weakness in the arms and later in the legs. She attributes her illness to overwork. Eighteen months ago, while singing, her tongue became paralysed and she could not articulate distinctly. Twelve months ago she had difficulty in swallowing and liquids came down her nose. Five months ago she began to have suffocative attacks. There is no history of diphtheria.

State.—There is slight double ptosis, the eye movements are sluggish and restricted in all directions, especially upwards. The right eye cannot be turned up at all and forced movements in any direction are attended with nystagmus; diplopia is present at times, the pupils and discs are normal. She could not close her eyes tightly and fibrillary tremor of the ocularis muscle occurred when she tried to do so. The tongue is weak, and speech indistinct. There is difficulty in swallowing; no paralysis of the soft palate could be discovered. The palatal reflex is present; mastication is impaired. The arms cannot be lifted from the shoulders, the deltoids are large and all the other muscles normally developed; there are no sensory changes, no

exaggeration of the reflexes and no electrical changes. The spinal muscles are weak, she is unable to sit up; when lying down she cannot get up at all without assistance; the legs are very weak, she cannot lift them off the bed. The gait is slightly waddling, and she would fall if not supported. The knee-jerks are a little increased, no ankle clonus, plantars absent. There are no sensory changes. The feet and hands are blue. The patient cannot lie down in bed, as she dreads an attack of suffocation. An attempt to examine the throat brings on inspiratory stridor and cyanosis to such an extent that the patient seems to suffocate. There are no hysterical symptoms. She remained under observation three months, when she was *in statu quo*.

Case 21, Suckling (15).—A female, aged 18, with a neurotic family history. Her illness is attributed to exhaustion, and commenced gradually three years ago. She is unable to walk more than a hundred yards without becoming exhausted. The arms are weak and the grasp feeble. She cannot hold her arms out or place them on her head. There is slight ptosis, squinting, and diplopia at times. The movements of the eyeballs are restricted in all directions. Nystagmus is present. The knee reflexes are a little increased, there is no ankle clonus. There are no electrical changes. She complains of difficulty in swallowing and mastication. After mastication the jaw drops, she has difficulty in articulation. She is slightly choreic, has no suffocative attacks, and there are no hysterical symptoms.

Case 22, Wernicke (16).—A female, aged 16, admitted June 16, 1893, with the following symptoms: Ophthalmoplegia externa, incomplete double ptosis, bilateral facial weakness, upper and lower. Paresis of motor fifth and of muscles of deglutition; only fluids can be swallowed and they occasionally come through the nose. No affection of the tongue or speech. Paresis to a marked degree of both neck and shoulder muscles. Upper arm, forearm and hand weak, but she can use them still. Thigh muscles slightly stiff, otherwise the legs are not affected. Her illness developed gradually four months ago with slight temporal headache. The electrical excitability of the muscles is intact although in a few muscles quantitatively slightly diminished. The tetanic induction stream produces instead of a lasting only a temporary muscular contraction. The doubtful diagnosis was amyotrophic lateral sclerosis with bulbar complications.

July 17, 1893.—She was admitted to Clinik of Professor

Mendel. She now has palate, tongue and jaw weakness, and anæsthesia of the pharynx and larynx.

Sixteen days after admission the patient died suddenly of asphyxia. The autopsy was negative, the only change being considerable vascular dilatation, particularly in the Hirnstan, and numerous recent hæmorrhages.

Case 23, Mayer (18).—A patient suffering from difficulty in speaking and swallowing, weakness of the jaws, bilateral ptosis, paresis of extremities, and abnormal fatigability; the symptoms were worse towards evening and after exertion. There was no atrophy and no disturbance of the reflexes. Temporary improvement was followed by sudden death by choking.

Post mortem.—It was found that the cells of the hypoglossal nucleus, with the exception of a small number which were vascuolated, were completely normal, as were the cells of the anterior horns and pyramidal tracts. On the contrary, there was a marked pathological change in the intra-medullary portion of the anterior roots, also in the hypoglossal root, particularly distinctly seen in Marchi preparations, such preparations showing all along the anterior roots, as also in places on the hypoglossal root, stripes and flakes of dark myelin. The intra-medullary parts of the anterior roots, as well as of the hypoglossal root, appear distinctly atrophic, when the nerve sheath stain (Markscheiden färbung) is used.

Case 24, Strümpell (24).—A female, a field-worker by occupation, aged 21. Admitted December 30, 1893. Ten months ago she began to have difficulty in speaking and in raising the upper eyelids; six months ago she noticed weakness in the arms after working in the fields, and in the legs after walking, also difficulty in swallowing and chewing, and double vision.

Present state.—There are bilateral ptosis, weakness of the face and jaw muscles, and weakness of the extremities without atrophy. Speech is indistinct, the letters *b*, *d*, *g*, *j*, *m*, *n*, *s*, are especially badly pronounced. Sensation and the sphincters are normal; the reflexes are normal and could not be exhausted, although after repeated tapping the knee-jerks seem to diminish slightly in activity, great exhaustion is rapidly produced and amounts to almost complete paralysis of nearly all the muscles. She was worse at the menstrual periods. From January, 1894, until she died she had almost daily severe attacks, during which, if walking, she would fall suddenly to the ground, become cyanosed and breathless. Respiration became noisy, sputum accumulated in the mouth,

she could neither spit it out nor swallow it; the tongue sank back; if in bed she would sit up with her head bent forwards, saliva flowing out of the mouth. The arms when raised fell flaccid by her side, the extremities were cold. She developed a catarrhal pneumonia, and died suddenly during one of these attacks.

A *post mortem* was made, the medullary nuclei being particularly examined, and no changes were found.

Case 25, Murri (25).—A patient suffering from severe bulbar symptoms, characterised by a tendency to improvement. The disease has lasted for ten years; the myasthenic reaction is present, and the early exhaustion after exercise. In this case, even when complete exhaustion was produced in the muscle by an undue current, the muscle can be readily contracted, voluntarily (and the reverse).

Case 26, Charcot and Marinesco (22).—A boy, aged 13, suffering from complete ophthalmoplegia externa, complete paralysis of the lower, incomplete of the upper, extremities, trunk and face. Death occurred after three months with bulbar phenomena. The symptoms developed in the order above mentioned. There is no affection of sensation nor of the sphincters, and no mental change. There is no affection of the internal organs except an enlargement of the thyroid gland. There are no qualitative electrical changes, but in some muscles there is diminished excitability both to the galvanic and faradic currents.

Post mortem.—Some recent hæmorrhages were found beside the aqueduct of Sylvius, and some others, but less pronounced, at the level of the ventral nucleus of the vagus. No other changes were met with in the central or peripheral nervous systems.

Case 27, Pineles (23).—A male, aged 25. No ætiological factors were discoverable. The onset of the disease was sudden, with headache, giddiness, diplopia, and right-sided ptosis.

In July, 1893, he had difficulty in chewing and swallowing, nasal and indistinct speech, weakness in the arms and legs, and attacks of dyspnœa and palpitation. All these symptoms developed within a few weeks, and were most marked towards evening. The mental state and sensation were normal; there were no hysterical stigmata. There were no electrical changes and no atrophy. The knee-jerks were somewhat active; the palate reflex was present.

Pineles records two other cases which ended fatally after one and a half and two years respectively, death being due in each case to an attack of dyspnœa.

Case 28, Collins (26).—A woman, aged 27, the mother of two healthy children. Three years ago, when six months pregnant, she complained of weakness of the extremities, and soon after left-sided ptosis and diplopia developed, the ptosis disappearing after two or three months. Ptosis of the other eye then appeared, lasting for two months. Following this there developed difficulty in articulating, fatigue on chewing food with difficulty in swallowing and in moving the tongue, with almost complete inability to move the extremities. On first attempting to talk speech is fairly distinct, but soon the tongue becomes heavy, the lips unwieldy, the voice hoarse, weak and nasal. Fluids regurgitate through the nose on swallowing. The weakness is sometimes greatest in the extremities, at others in the muscles supplied by the cranial nerves. The intrinsic muscles of the eye are normal. Attacks of distressing tachycardia and paroxysmal dyspnœa occurred.

These symptoms improved, but she relapsed three months ago. She wakened one morning with paralysis of one side of the face which lasted three days. Since then gradual improvement has occurred. She can now walk provided she does not overtax her strength. She cannot tightly squeeze her eyelids together, she cannot hold her lips firmly together, the tongue is weak (not atrophic), the voice nasal, labials are imperfectly pronounced; the arms easily tire.

The reflexes at first appear normal, but after striking the patellar tendon several times the response gets less and less, finally ceasing to reappear after rest. There is no reaction of degeneration. The electrical responses are normal. There are no fibrillary twitchings.

Case 29, Fajersztajn (27).—A man, aged 23, was seen in April, 1895. He was said to have caught cold ten days previously. This led to great weakness in the legs and difficulty in chewing, swallowing and speaking.

Present condition.—Slight ptosis. The patient tries to prevent this by over-action of the occipito-frontalis. Blinking does not occur often; there is slight lateral nystagmus on looking to the lateral extremes, the orbicularis palpebrarum is weak, especially after frequent closing of the eyes. He can whistle for a few seconds; he can only blow out light two or three times; occasional slight fibrillary twitching of mentalis occurs; the tongue easily gets tired; the pharyngeal reflex present; there is decided weakness of the masticatory muscles increasing with their use. Biting crusts causes great fatigue. There is difficulty in swallowing; the cervical muscles are weak, but the head is held

erect. Speech is nasal, weak and rough, and becomes more nasal and weaker when continued; after counting forty to fifty slowly, he becomes aphonic. The breathing is chiefly diaphragmatic and is quickened by the slightest effort; there is decided weakness of the trunk muscles, and he cannot raise himself into the sitting posture while in bed without using his arms; he can only lift his arm over his head ten to fifteen times, even lifting a spoon to his mouth soon tires him; he can write twenty to thirty letters with a steady certain hand, when the writing becomes irregular and soon impossible. There is similar weakness in the lower extremities; he is obliged to rest after walking a few paces. The plantar, cremasteric and abdominal reflexes are normal; the tendon-jerks and electrical reactions are normal.

After six months' rest in bed and administration of strychnine, improvement in all the symptoms followed; afterwards at times there were transient relapses The symptoms were always worst towards the end of the day.

Case 30, Silbermark (28).—A male, a cook, aged 34, entered Nothnagel's Clinik, June, 1896; his family history and previous health were satisfactory. In December, 1894, he found when rolling dough that the third and fourth fingers were weak and tended to drop. After a short improvement, weakness and a feeling of fatigue appeared in the upper limbs, a few months later in the lower. Difficulty in chewing, and regurgitation through the nose appeared, together with facial weakness, difficulty in swallowing and nasal speech. Great variations occurred in the intensity of the symptoms. He always felt better in dry, warm weather than when it was cold and damp.

State.—June 5, 1896. A slight degree of ptosis is present. The ocular movements are free; von Graefe's symptom is present; the pupils react well. There is no weakness of the jaw muscles. The facial weakness is most marked on the left side. There is no weakness of neck; the limbs are affected. Tiring of one hand had no effect on the other.

Case 31, Devie et Roux (29).—An unmarried woman, aged 43, entered hospital July 3, 1894. The symptoms gradually developed two to three months ago without any affection of her general health. She has almost complete paralysis of the extensors and rotators of the head and neck: the head falls forwards, the chin touching the chest; she can only with great difficulty lift the head without using her hands. The rotators

are somewhat less affected. Beyond slight paralysis of the right inferior facial there are no bulbar symptoms. Bilateral ptosis is present, very marked on the left, much less on right side; the globes are practically immobile; the orbicularis palpebrarum is intact; the reflexes are normal; fundus: there is slight perivascular œdema of the optic discs, the margins being indistinct. Potassium iodide and mercury were used.

July 16.—The cervical muscles are less paralysed, also the ocular muscles, but the ptosis remains unaltered. Specific treatment steadily pushed.

August 10.—There is steady improvement, and greatly increased power in the cervical muscles. The muscles of eyeball are almost normal; the ptosis is very slight in the morning, but gets marked by mid-day.

August 20.—Movements of the head are normal, the ocular movements almost so. The ptosis is confined to the left eye and this only towards the end of the day; there is slight nystagmus. The mercury was stopped, iodide continued.

September 16.—Left the hospital practically well.

December.—Re-admitted for observation. Iodide has been continued. She has slight and transitory ptosis, but no other symptoms are observed. Discharged May, 1895.

July 31, 1895.—The patient was seen on this date, practically well.

November 20.—Third admission. Ptosis for two months reappeared on the left side, with slight diplopia, most marked towards the end of the day. Except for slight paralysis of right face, there are no other symptoms. There is no atrophy of the cervical muscles.

Case 32, Kojewnikov (30).—A male, aged 54, who had previously been healthy. There is no history of syphilis or alcoholic excess. The disease began suddenly some hours after a severe chill, when he first noticed weakness of the lips. Soon after he had a feeling of tightness of the face, difficulty in closing the eyes, in chewing, in swallowing, and nasal speech.

State.—The patient suffers at present from leftsided ptosis, weakness both of the upper and lower face, at times diplopia, limitation of the tongue movements, difficulty in swallowing (he had to be fed with a tube). He becomes easily tired when speaking, breathing is laboured, the face muscles are manifestly atrophied, both masseters, particularly the left, being very atrophic. The skin and deep reflexes are normal and there is no affection of sensation or of the special senses. At first he

became worse, then he improved. Six months later after a chill the symptoms returned. There was then a slight difference between the pupils and slight insufficiency of the left internal rectus. The mouth was usually held half open and there was great tiredness of the muscles in chewing, speaking and swallowing. There were no electrical changes. The patient died suddenly, probably from paralysis of respiration, two and a half years after the onset of the disease. There was no autopsy.

Case 33, Kojewnikov (30).—A female, aged 17. No history of syphilis or alcohol. A brother has spasmodic asthma. Since the onset of menstruation in 1889 her health has been indifferent. Her present illness seems to have dated from this. She first noticed a difficulty in utterance and nasal voice. This continued till February, 1890, when she could neither whistle, blow out a candle nor expectorate, and was suffering from ptosis and difficulty of swallowing.

September, 1894.—There is weakness of the facial muscles, especially the lips; the lower lip falls. Speech is difficult and easily tires; the voice is nasal; there is left-sided ptosis. There is slight symmetrical wasting of the tongue. The soft palate is immobile. The muscles of the shoulder and hip are weak. Sensibility and the tendon reflexes are normal.

Summer, 1895.—There is defective taste and occasional diplopia.

September.—She is much worse; there is weakness of the extremities, an inclination inwards of both eyes, and difficulty in holding the head up—it falls forwards.

October.—Speech is worse, swallowing impossible.

October 14.—The right pupil is slightly the larger, she cannot firmly close the eyes; mastication is very difficult. There is no atrophy of facial or masticatory muscles, but distinct atrophy of the tongue. The soft palate is almost immobile; defective adduction of the cords is seen on phonation; the posterior cervical muscles are weak and somewhat atrophied.

Electrical reaction; facial muscles show slight quantitative diminution to electrical currents. The soft palate does not respond to either current. In the tongue muscles A.C.C. is slightly better than K.C.C.

There is a distinct trace of sugar in urine; the patient has not menstruated since April.

Sensation is normal. She can only taste salt, and that imperfectly.

She is low spirited and very irritable.

The gravity of the symptoms varies, the muscular weakness being least marked in the morning.

October 26.—An attack of dyspnœa occurred during sleep at night, she was unconscious and cyanosed; it lasted three hours. She has had similar slighter attacks since.

November 1.—She improved from this date; sugar permanently disappeared from the urine on November 13.

December.—*In statu quo.*

In April, 1896, she left the hospital, still complaining of the chief symptoms.

Case 34, Toby Cohn (31).—A girl, aged 19, who has been ill for a year with weakness and tiredness in the arms and legs, ptosis, difficulty in swallowing, regurgitation of fluids through the nose, and nasal speech. All her symptoms are more marked towards evening.

On examination she was found to have double ptosis and lagophthalmos on both sides, a fixed expression with facial weakness on both sides; the jaw movements are feeble, the palate easily tired, speech is often nasal and difficult to understand, the letters *g* and *k* being especially difficult to pronounce. The tongue is normal, the neck muscles easily fatigued. The arms too are easily tired, and cannot be raised so well when sitting as when standing or lying. A similar condition of the lower extremities exists; after walking for some time a waddling gait develops as in the dystrophies. The knee-jerks are pretty lively. A paresis of the abdominal muscles exists. There is no ataxy, and sensibility is everywhere normal. The myasthenic reaction is very distinct; there are no other electrical changes. The internal organs are sound, the urine healthy, and there is no mental change. The family history is good; she had no preceding illnesses. An abnormal increase of the leucocytes is present in normal proportions.

Case 35, Kalischer (32).—A male, aged 64. No history of syphilis or alcohol. He had small-pox forty years ago, but otherwise his health has been good. In September, 1892, he developed double ptosis, paralysis of the extrinsic ocular muscles, and a week or two later paresis of all four extremities. A flaccid paralysis. The deep reflexes are increased, the skin reflexes lost; there is no atrophy or fibrillary tremor; the extensors are weaker than the flexors. There is no reaction of degeneration, but some diminution of faradic excitability in muscles and nerves.

There is no sensory disturbance, no sphincter trouble. The paralysis is always least in the morning. Later on the pupils

became sluggish, particularly to light. Death occurred apparently from paralysis of respiration.

Post mortem.—Microscopical examination. The changes observed in the microscopical examination were numerous old and recent hæmorrhages, some large, some small, in the whole of the cerebral gray matter from the posterior part of the third nucleus to the sacral region.

There was degeneration in the nuclei and cells of the anterior horn all through the medulla and cord, varying in degree in different parts.

The position of the lesion did not correspond with the symptoms present during life.

Case 36, Widal et Marinesco (33).—A man, aged 31. Suspected of having tuberculosis. He developed severe headache, and in a few days the typical features of asthenic bulbar paralysis—ptosis, diplopia, weakness of external eye muscles, deep monotone voice, facial and palatal paresis, difficulty in chewing and swallowing, weakness in the neck and arm muscles, and the legs were easily fatigued. Typical variations in the intensity of the symptoms occurred. Dyspnœa and a small fast pulse were present. He died suddenly fourteen days after the commencement. For the last eight days the fever was 38-39°, and there was a trace of albumen in urine.

Post mortem.—The third, sixth, seventh and twelfth nuclei and cells of anterior horns examined by Nissl showed *chromatolysis*, the vessels were widened, there were no hæmorrhages; in the roots of the third, seventh and twelfth nerves degenerated fibres were seen by the Marchi method.

Case 37, Berkeley (34).—A male, aged 28, whose family history was good. When 14 years old slowness of movements attracted attention. His sister said, "When he stooped he never seemed able to get up again." Seven years ago the symptoms of his present illness were first noticed; he was easily tired and rather somnolent; he had an inordinate appetite. Four years ago he was troubled with vomiting after meals, which lasted for three or four months. Soon after he developed pronounced *deafness*, which persisted for several months, and has never entirely disappeared. His gait was clumsy and he was apathetic. Articulation was difficult and gradually became worse. A year ago the eyeballs became "stiff," and double ptosis with a peculiar facial expression appeared. There was no history of syphilis or alcoholic abuse.

State.—September, 1892. The facial expression is dull and heavy, the patient usually sits with his head bent forward because of weakness of the muscles at the back of his neck. The muscles of extremities are very feeble and easily exhausted. There is no atrophy or fibrillary twitchings. Sensation is normal; he is somewhat deaf. Bilateral anosmia is present. The pupils are semi-dilated and do not respond to light or accommodation. Deep reflexes increased, muscles react normally to faradism but are easily exhausted. Heart regular. Pulse fast (120). No albumen or sugar in urine. Incomplete double ptosis exists; the internal and inferior external rectus and oblique muscles of both eyes are paralysed. The eyes are practically immobile except that he can roll them upwards slightly. The masseters are weak and easily fatigued by faradism. There is weakness of the face muscles, the forehead is smooth, the orbicularis responds feebly, whistling is impossible, all the letters of the alphabet badly enunciated, the palate hangs low, the pharyngeal and palatal reflexes are well marked; no laryngeal examination was made. The tongue movements are defective. Contented disposition. Duration of illness ten years.

July 13, 1895.—While being fed with soup he *suddenly* choked and *died from asphyxia*; he had had no previous attack of dyspnœa.

Post mortem.—An old hæmorrhage was found in corpus callosum, having occurred probably in early life, perhaps when he had variola, aged 18 months.

This might account for the cerebral condition. No changes were present in the nuclei of medulla or pons, either in the cells or vessels; particular attention paid to third nucleus, but nothing abnormal found.

Case 38, Schlesinger (35). (*Very doubtful case.*)—A female, aged 29, admitted August 12, 1894, had had good health. Six weeks previously she became suddenly ill and experienced great difficulty in swallowing. Fluids especially were difficult to swallow, regurgitation through the nose took place, and there was a copious secretion of saliva which frequently flowed out of her mouth. There was frequent vomiting. After a few days she was better, but the arms and legs began to feel weak. Her condition gradually got worse.

State.—August 19, 1894. The mental state is normal, speech is slow and hesitating, the pupils are equal and react promptly. The eye movements are free, there is no diplopia, and no affection of the face, tongue, or larynx.

A high degree of general muscular atrophy is present. The

muscles in shoulder region on both sides are both fairly markedly atrophied, particularly the posterior part of deltoid.

There is marked atrophy of the thenar and hypothenar eminences.

The arm-jerks on the right side are absent, on the left side they are very feeble. The muscles of lower extremities are much atrophied, but react to faradism. The knee-jerks are obtainable, but not increased, and sensation is completely normal except a large patch corresponding to the deltoid and right side muscles, where the sense of temperature is lost. There is retention of urine, requiring the catheter, and obstinate constipation.

Towards the end of August she became worse, vomiting and difficulty in swallowing developed, with increased salivation and rapid pulse. Tongue deviated to the left; the larynx was normal.

September 18.—Died from paralysis of the diaphragm.

Post mortem.—Nothing abnormal found; Weigert-Pal, Nissl, and Marchi all being used.

Case 39, Brissaud et Lantzenberg (36). (*Doubtful case.*)—A carpenter, aged 41, who had had bronchitis and pleurisy, noticed great general wasting after a cardiac illness (? myocarditis). One and three quarter years after the onset he had right facial weakness with inability to open the eyes to the full extent, a wasted, powerless tongue, double vision and weakness of the external eye muscles. Atrophy and weakness of sterno-mastoid and neck muscles, also in the arms and legs, with occasional fibrillary twitchings. Diminished knee-jerks, rough nasal speech and dry cough. Six months later there was a distinct improvement in most of his symptoms, except the fatigability of the muscles. No affection of swallowing. The jaw muscles, which were weak, now act well. No myasthenic reaction. Progressive improvement occurred. No statements as to ultimate result.

Case 40, Karplus (37).—A female, aged 24. Healthy family. When 5 years old she developed slight right-sided ptosis, which progressed. The following week the left lid was also affected. After a year the ptosis gradually disappeared. Since then every year for periods of several weeks ptosis has appeared, worse towards evening. There was occasional diplopia.

November, 1894.—She had ptosis, paralysis of the external eye muscles, weakness and tiredness of the upper and lower extremities.

December, 1894.—Paralysis of the external eye muscles. Paresis of the frontalis and ocularis; weakness in mastication.

After two months the weakness of the extremities disappeared and the ocular movements improved.

Autumn, 1896.—No ptosis; weakness of superior and inferior recti is present.

Case 41, Eulenberg (38).—A man, aged 28. Family history satisfactory. He had inflammation of the throat with a diphtheritic-like membrane a few months before onset of present illness; no syphilis; no excessive alcohol or tobacco history. In December, 1894, he saw double, and complete right-sided ptosis developed suddenly; later ptosis developed on the left side. In February, 1895, these symptoms disappeared, to reappear again gradually in November of 1895. From the middle of 1896 to the middle of 1897, he was again quite well. Since June, 1897, he has suffered from irregular cardiac action, ophthalmoplegia developed, and weakness of both arms and legs, especially on the right side.

State.—December 6, 1897. Complete ophthalmoplegia externa on both sides, the muscles are not all affected to the same degree. The right pupil is dilated and its reactions are sluggish; the papilla is of a whitish colour, but there is no disturbance of function (later the dilated pupils scarcely reacted at all to light); on the left side the optic nerve was hyperæmic; double facial weakness, particularly of the orbicularis palpebrarum muscle; movements of the jaw and palate are weak and deglutition is difficult; on the left side his ability to hear deep tones is diminished (insufficiency of the tensor tympani?). There is weakness of the trunk and limb muscles which varies from time to time. There is striking increase of the muscular sensibility (*auffallende steigerung der muskelsensibilität*) in the limbs for electricity, especially for faradic currents. The myasthenic reaction is present in the extensors of the left forearm, the right extensor indices, and markedly in the interossei of the right side. Great improvement occurs after prolonged rest. There is no muscular atrophy; the reflexes are preserved; sensibility is intact.

Case 42, Mailhouse (39).—A male, aged $2\frac{3}{4}$ years. He has suffered greatly from the heat. A week ago he had a mild attack of diarrhœa a few days prior to the onset of the first symptoms.

July 6.—The child could not hold up its head properly, at other times the head was as erect as usual. There was no fever or vomiting, and no complaint of pain. He was unable to chew or to swallow properly, fluids tended to regurgitate through the nose. At times speech was affected so that he could not be clearly understood, at other times it was natural. He has not been con-

fined to bed. The parents noticed that his eyes were half closed and that he could not smile. The family history was good.

State.—Marked bilateral ptosis, pupils and external eye muscles normal.

The face is expressionless, he does not smile, saliva runs out of the mouth. The mouth is half open, the soft palate normal, and the voice good. The reflexes are normal. After walking a short way he becomes unsteady on his legs, so that he asks to lie down. There is no atrophy. The electrical examination was unsatisfactory owing to uneasiness of child. The pulse 84; temperature normal. He was ordered to rest in bed and given strychnine and an intestinal antiseptic.

July 19.—Mailhouse was sent for because of peculiar rolling of the eyes; his mother thought he was weaker.

July 31.—Ptosis is gone; there is no strabismus; he can chew, swallow and smile, and runs about as he did before he was taken ill.

August 4 (*evening*).—A choking attack occurred during an attempt to swallow; weakness of neck muscles and regurgitation of liquids, &c., followed. He was better by the time the doctor got there.

August 5.—He has been up and down during day. While attempting to drink coffee from a cup a little difficulty in swallowing was noticed, his head drooped and he was dead.

He never had any attacks of dyspnœa; the heart's action was good.

There was no autopsy.

Case 43, Laquer (40).—A female, aged 21, whose mother died of phthisis. She had suffered previously from anæmia. She developed right-sided ptosis and double vision, and later difficulty in swallowing and regurgitation through the nose; ptyalism was present. Speech became indistinct and nasal; the arms and legs became weak. Three months before she had given birth to a child.

State on examination.—Left facial paresis; left-sided ptosis; weakness of the left internal and right external rectus, and diplopia; difficulty in whistling; speech slow, very indistinct and nasal in character; larynx normal. Tenderness on pressure over the fifth nerve on both sides. The knee-jerks were increased; there was shortness of breath. Great weakness in arms and legs was present, and often great difficulty in swallowing. The symptoms varied in intensity. The patient died suddenly one and a half years after the onset of her illness. The autopsy was negative, but there was no microscopic examination.

Case 44, Laquer (40).—A male, aged 49. Family history good. He has had pneumonia and repeated attacks of gout. He complained of pain and weakness in the right arm and leg; later, weakness in both arms. Double vision, ptosis, tiredness of the eye, face, and jaw muscles appeared; also of arms and legs. Fatigue of one group of muscles produced exhaustion in others, the myasthenic reaction was demonstrated in the biceps; there was no atrophy or bladder trouble, while sensation and the reflexes were normal. After an attack of bronchitis all the symptoms became intensified. His expression was that of a man falling asleep after the mid-day meal. Chewing, swallowing, and speech all showed early signs of fatigue. After glancing outwards and inwards several times the eyeball became fixed. There was no laryngeal affection.

Six months after its onset all the symptoms of the disease became much more pronounced. Distinct atrophy of the deltoid, biceps, triceps, pectorals, interossei, quadriceps, and calf muscles, developed. The tendon reflexes were brisk and could not be tired out. The myasthenic reaction was present. Some muscles, especially the gastrocnemii and extensors of the forearm, are tender on pressure.

Case 45, Wheaton (41).—Governess, aged 33, who had always been nervous. There was no neuropathic heredity. She had worked very hard. About the second week in July, 1896, she had a tooth extracted; the dentist told her that she must be very careful, or her gums would start to bleed and she might bleed to death. This frightened and worried her greatly. On July 21 she had a severe headache, in the morning she could raise the eyelids only with difficulty and the light pained her. On July 22, excepting for these signs and in addition ptosis and a slight increase of the reflexes, nothing abnormal was noted. During the next few days the ptosis became more marked, there was difficulty in chewing and in keeping the mouth closed. She was unable to sleep and had occasional attacks of palpitation. All the symptoms were increased by exertion.

August 14.—She had difficulty in walking, the muscles of the the neck were weak, menstruation began, and all symptoms increased in severity. She had difficulty in swallowing.

August 31.—She had a severe choking spell.

September 7.—To-day the patient had a bad spell of difficulty in breathing.

September 14.—She died in an attack of dyspnœa.

Mentally somewhat despondent. The reflexes increased

gradually during the disease; there was no atrophy of the tongue and no tremors. The pulse was usually between 90 and 110.

There was no autopsy.

Case 46, Saenger (43).—A female, aged 22, seen December 29, 1896. She has been married one and a half years but has no children. Her previous health was good. Four weeks ago, she suffered from headache and sore throat. Difficulty in swallowing and double vision followed. Eye disturbance was present before the sore throat. The patient is anæmic and has a fixed facial expression. She has difficulty in preventing the upper lids from drooping. The eyes cannot be moved either inwards or outwards. The pupils are equal and react normally; she has difficulty in swallowing. The palate is weak. The voice is feeble. The pulse is somewhat rapid and regular. There is great diminution of power in both the upper and lower extremities; the gait is normal, but walking soon makes her tired. Now and again there is weakness of the neck muscles; the symptoms are more intense towards evening. Sensibility and the reflexes are intact; neither the myasthenic reaction nor reaction of degeneration are present.

January 1, 1897.—The patient is somewhat better, she still has pain in the spine and nape of the neck.

January 10.—She walks better.

January 13.—There is marked diplopia.

Subsequently she became pregnant and got practically well of the paralysis.

Case 47, Montesano (45).—A shoemaker, aged 17. Fourteen days after a cold bath, which he took when overheated, he was affected with weakness in the legs, so that walking was soon impossible; then fatigue appeared in the arms and trunk, and diplopia. The spleen was slightly enlarged. The muscles were very easily fatigued. There was inability to dorsiflex the hand, and to extend the fingers, deviation of the tongue toward the left and weakness of the left side of the face. The superior oblique and the external rectus of the left eye were paralysed. There is no affection of sensation. The faradic excitability of the muscles of the left half of tongue and right hypothenar eminence is diminished.

The myasthenic reaction is present in the lower extremities, especially in the quadriceps. Voluntary movement is still possible after exhaustion by faradism.

After four months the patient had recovered, excepting for slight diplopia and slight weakness in the finger movements.

The author suggests an organismal origin for the disease, the organisms becoming virulent in consequence of the above-mentioned chill.

Case 48, Kostetzki (47).—A male, aged 24, who had suffered from attacks of palpitation and dyspnœa, developed weakness in his trunk and extremities, so that he had to give up work. The voice became nasal, the breathing was uneasy. The symptoms gradually disappeared after a few weeks. Two months later, after a severe mental shock, difficulty in swallowing developed.

Present state.—The patient is very thin, his muscularity is feeble. There is bilateral ptosis, and the expression is mask-like. The muscles of the face are paretic. Movements are scarcely noticeable. The tongue can only be protruded very slightly. The pharynx and larynx muscles are paretic. Speech is indistinct and nasal; chewing is difficult, and he has difficulty in swallowing and breathing. There is paresis of diaphragm. Attacks of palpitation occur. Paresis and atrophy are present in various muscles of trunk and limbs—the pectoral, deltoids, thenar and hypothenar muscles, glutei and spinal muscles. The reflexes and sensibility are unaffected. The muscles are quickly fatigued (for example, diplopia develops after a few movements of the eyes). Improvement occurred after several weeks' treatment; a relapse then followed, and this variation in the symptoms recurred several times.

The author is of opinion that this case forms a transition variety between chronic bulbar paralysis and the so-called asthenic bulbar paralysis.

Case 49, Angelini (49). (*Doubtful case.*)—A man, aged 42, who had been nervous from youth. The illness commenced after emotional excitement, with tiredness of the muscles of the eye, the limbs and jaws. He also became soon fatigued after writing, speaking or reading, so that after a few words he could not remember what he had said. He had a severe feeling of pressure over the head and a feeling of stupor. After a short pause he could go on with his business. Sensation and the reflexes were normal. The myasthenic reaction was not present. Under hydrotherapy and strychnine the symptoms disappeared. Two years later there was a slight relapse from which he soon recovered.

The clinical picture shows an undoubted resemblance to myasthenia gravis. But while in this disease the symptoms are confined to the motor apparatus, in the case above described the higher functions were affected.

The case is more probably one of hysteria than myasthenia gravis (Angelini).

Case 50, Harry Campbell (53).—A governess, aged 28. Seen December, 1898. Twelve months ago she noticed she could sing in the morning but not at night, and since then she has gradually developed the symptoms she now complains of, *i.e.*, difficulty in swallowing; sometimes solids "stick in the throat," and fluids pass through the nose; nasal speech; a tired feeling at the back of the tongue. She cannot whistle, nor empty a spoon with the upper lip. There is upper facial weakness, but the corrugator supercilii acts well; great weakness of the orbiculares palpebrarum; after eating for some time the food tends to accumulate between the gums and teeth; mask-like expression; weakness of the face, soft palate and tongue. The above symptoms are all worse after using the affected muscles for some time. They are thus much better in the early morning than towards evening. No ptosis or diplopia. Knee-jerks and electrical reactions normal.

Later she complained of weakness in the arms, especially the right one; this she noticed in lifting her arms "to do her hair." This difficulty lasted a short time only and did not recur.

January 27, 1899.—All the symptoms much worse; she can scarcely close her eyes.

February 10.—Much better.

April 29.—Improvement continued until April 7; during last fortnight has been worse. Has had slight diplopia.

February 16, 1900.—The symptoms have fluctuated in severity since the last note was made, but the general tendency has been improvement.

Case 51, Sinkler (50).—A married woman, aged 37. Two brothers and a son suffered from nystagmus and rotatory head tremor, another son has nystagmus. There was no suspicion of syphilis. She had typhoid fever seven years ago, followed by bilateral ptosis, which lasted for four or five months. Two years ago she had diplopia. One year ago she was confined, the labour was a difficult one. A few weeks later swallowing and speaking became difficult, and weakness in the arms and legs developed. Since then at each menstrual period the symptoms have been worse.

State.—The face is mask-like, but there is no paralysis of the facial muscles. She can show her teeth and whistle. Speech is nasal and indistinct. After speaking for some time complete

aphonia results. Deglutition is difficult, sometimes regurgitation occurs through the nose; the jaws soon become tired with chewing, as do the arms and legs after exercise, the gait becoming unsteady and shuffling. The knee-jerks are present and apparently do not become exhausted on repeated testing. All the muscles respond to faradism and the faradic irritability is not exhausted. There is no affection of sensation and no headache. There is partial double ptosis, the pupils react normally, almost complete paralysis in left external rectus, and partial of the left superior rectus exist. Patient is of a cheerful disposition. The fatigue which occurs in the eyes, throat, legs and arms is very characteristic.

Case 52, Punton (51).—A married woman, aged 25. She suffered from rheumatism before marriage. She has been ill for three years. Her only child was born just previous to the first symptoms; the labour was prolonged and difficult. She has never been well since delivery.

Diplopia, dimness of vision, and left-sided ptosis were the first symptoms. Mastication, articulation, and swallowing became difficult, voice became nasal, the tongue was less dextrous and felt numb. Weariness, prostration, and rapid heart's action followed slight exertion. She became unable to whistle, and strabismus appeared in both eyes. She developed a continuous lachrymation, and lost two stones in weight.

State.—The eyeballs protrude slightly and cannot be moved in any direction. She complains of excessive lachrymation during the waking hours. Double ptosis is present, left greater than right. She has diplopia; the facial expression suggests a putty-like blankness. Profound paresis of the face, right greater than left; she cannot whistle. The masseters are equally paretic, the bite is feeble, there is no atrophy, tremor or twitchings. The tongue is thick, flabby, and completely devoid of lateral movement. The palate moves very little. Speech is thick, indistinct and nasal, and deglutition of solids difficult, while fluids regurgitate through the nose. The left vocal cord is paralysed, the power in the extremities is normal; there is no atrophy, and no twitchings or sensory symptoms; the superficial reflexes are normal, the deep are slightly exaggerated. There is no ankle clonus; electrical reactions are normal; urinary analysis negative. The pulse 90, regular.

Case 53, Clifford Allbutt (52).—A girl, aged 18. The personal and family history are good. She caught cold, followed by stiffness of the tongue and jaws, which disappeared after a few weeks.

Six months later she was seen by Clifford Allbutt. She spoke oddly, "as if with a potato in her mouth;" when she tried to read a book she began well, but soon the disorder set in and she became unintelligible. Her friends said that she read much better when at home quietly. A few weeks before she had begun to have difficulty in swallowing. "She choked over her meals, more when she was tired." There was no evidence of hysteria or of organic disease observed, except that the forefinger of the left hand seemed weak and fumbling. She moves her tongue and jaws and all the muscles of the face, throat and orbit quite normally. The palatal reflex was normal. A provisional diagnosis of hysteria was made. Under treatment she improved very much. One evening the symptoms became worse, especially the swallowing, and her mother spoke to her somewhat sharply. Next morning she went to her mother's room to protest, when she suddenly fell to the ground, was convulsed, turned blue and died. There was no autopsy.

Case 54, Seiffer (54).—A female, who has been ill eight years (age not given). The illness came on without known cause. Five years ago the fatigue in the arms and legs, which was present before, became more marked, her gait became waddling, jaw weakness and ptosis developed. The tiredness was always more marked towards evening.

State.—She has double-sided ptosis; the eye muscles act normally, but at times nystagmoid movements are present. The patient quickly becomes tired on trying to count aloud, ultimately she is quite unable to do so. She can only raise herself with difficulty from the horizontal position. The arms if held up quickly become tired, as do the legs after walking, when she wavers and ultimately staggers. There is a distinct myasthenic reaction; nothing peculiar as regards the blood is present.

Case 55, Buzzard (55).—A schoolmaster, aged 40. His paternal uncle died in a lunatic asylum, a paternal aunt died of creeping paralysis; there is no history of syphilis or alcohol. Seven years ago, after taking cold baths, the little and ring fingers of the right hand felt weak. This recurred from time to time; nasal voice, feeling of constriction in the throat and ptosis followed. During the year 1895, excepting the weakness in the right index finger, he had no trouble; legs and arms then became weak, and later there followed diplopia, weakness in the tongue and jaws, difficulty in coughing and in masticating, attacks of dyspnœa and palpitation, fatigue; cold weather and mental excitement all increase the intensity of his symptoms.

On admission.—Very intelligent man, indistinct nasal speech, diplopia, right-sided ptosis, upward movement of eyes defective, both external recti weak, pupils react perfectly, jaws easily tired, double facial weakness (especially upper face), tongue moves freely but easily exhausted, palate movements and reflex power poor. After repeated deep respirations the glottis finally opened less than at first. Great weakness of the neck muscles, arms and legs are weak, he has difficulty in dressing himself and cannot walk more than half a mile, no muscular atrophy, no fibrillary twitchings, myasthenic reaction present, no impairment of sensation, no sphincter trouble, arm-jerks poor, knee-jerks active, not exhausted by frequent tapping, nothing abnormal in the urine, other organs healthy.

While in hospital patient improved greatly, but a relapse occurred without apparent cause, the relapse characterised by the preponderance of severity of symptoms in the trunk and muscles of the extremities rather than those of the bulk.

Case 56, Buzzard (55).—A female, aged 24, housemaid. Rheumatic family history; no nervous disease. Anæmic for eighteen months, otherwise good health until a year ago. First symptom was giving way of the legs when walking upstairs, legs have been weak ever since; soon after arms became weak, speech became nasal, ptosis developed, diplopia, difficulty in mastication, in coughing and in swallowing were noticed; no attacks of dyspnœa or palpitation. When excited, symptoms are intensified; cold does not affect her; the symptoms are no worse at the menstrual period. On admission, no alteration in mental state, no affection of cranial nerves, speech almost normal at first, after reading aloud soon becomes indistinct and nasal.

Double ptosis, left pupil greater than right, ocular movements good, slight nystagmoid movements on extreme lateral deviation, convergence poor, orbicularis palpebrarum very weak, power of mastication very poor, so feeble that she cannot hurt a finger introduced into the mouth when she tries to bite it, tongue and palate movements fairly free but soon exhausted, extremities weak, no atrophy, no fibrillary tremors, myasthenic reaction demonstrated in biceps, sensation and sphincters unaffected, deep reflexes active. Knee-jerks cannot be exhausted by repeated tapping.

Case 57, Feinberg (57).—A man, aged 44, with no neuropathic heredity, developed obstinate constipation, with symptoms of obstruction, colic, meteorism and vomiting. On the fifth day he

had severe pain in occiput and region of upper dorsal spine, followed by numbness and weakness in the extremities, double ptosis, immobility of the eyeballs, feeble power of movement in the tongue and lips, inability to wrinkle the forehead, indistinct speech, continuous salivation, difficulty in chewing and swallowing, the latter only to a slight degree. The patient soon began to improve quickly.

State a month after appearance of symptoms.—A strong man. He has slight double ptosis, the ocular movements were slow, the up and down movements being defective, there was no nystagmus, the pupils react normally. The right side of the face is paretic, the left normal except for slight weakness in the left frontalis. Attempts at whistling and showing the teeth gave poor results. Speech is distinct with the exception of the labials, the tongue moves well, the power of biting is feeble on the left side. The palate moves well, the reflex is normal. There is no increase in the rate of the pulse or respiration. The occipital headache and pain in the back have long ago disappeared. All the other organs are normal; the urine contains nothing abnormal. The patient complains of great weakness and muscular prostration. There is no muscular atrophy and no disturbance of sensation. The knee-jerks are increased. There is no ankle clonus and no rigidity. He can make all movements, there is no ataxy. A feeling of tiredness is present even when he lies in bed, which is exaggerated when he stands or walks. There are no electrical changes.

Six weeks from its onset nearly all symptoms of illness had disappeared; since then he has remained well (a year).

Case 58, Leonard Guthrie (56).—Waitress, aged 23. Her previous health and family history were satisfactory.

Her illness commenced two years ago with difficulty in speaking; later, difficulty in swallowing, nasal regurgitation, inability to close the eyes and weakness in the hands appeared; all the symptoms were worse after exertion or excitement.

State.—A delicate-looking girl, but not markedly anæmic. Paresis of the orbicularis oculi and oris, tongue, soft palate, and probably of the pharynx. There is upper facial weakness but no ptosis, the ocular movements are perfect, there is no nystagmus. The pupils react well. She cannot blow out her cheeks or whistle; speech is indistinct and nasal. The vocal cords act normally. Deglutition is difficult; the jaw muscles are weak. She has fair power in the extremities, but soon becomes exhausted. Sensation and the sphincters are normal. The

myasthenic reaction is present. There is no muscular atrophy. After five weeks she was much improved.

Case 59, Raymond (59).—Woman, aged 25. Entered February 7. Dead child in October last.

January 8.—Took cold, was seized with paralysis of left infra-facial same evening, but Raymond is not certain whether the right facial escaped.

January 25.—Ptosis supervened (left); eight days ago right ptosis—this less. This paralysis is intermittent and almost disappears at times. Ptosis worse second part of day.

Towards February 7, complained a little of diplopia with difficulty in mastication. This fatigues her, has to stop every few "chews," due largely to paralysis of masseters, especially left; difficulty of protruding tongue, applying it to palate, and speech is *trainante;* labials ill pronounced; after speaking short time speech gets thick and less distinct. Intellect perfect; speech slow. Pupil reactions normal. Fundus normal. No sensory trouble. Reflexes normal. Patient complains of getting tired easily, and of not being able to walk for long.

Treatment.—Faradisation of paralysed muscles, sulphur baths and cold douches.

Improvement from February 26. April 15, cure complete.

Pflüger (gives no reference) (*Gazette des Hôp.*, 1,168), has cited a case of ophthalmoplegia externa consequent to menstrual trouble. (*De Graefe*, Landsberg, Schœler.)

Case 60, Harry Campbell (unpublished).—A girl, aged 19, who after an attack of scarlet fever three and a half years ago became paralysed all over (she could not even turn in bed). During the following year she gradually improved, and recovered power all over excepting in the face, which felt stiff. About twelve months ago she gave birth to a child. During pregnancy the face improved, but became worse again during lactation, which lasted for seven months.

On admission her chief complaint was a constant running of water from the eyes.

There is marked double facial weakness affecting both the upper and lower face; great weakness of the orbicularis oris and palpebrarum; occasional diplopia, and the eyelids often droop towards evening. There is no affection of the soft palate, tongue, or larynx, and no weakness of the extremities. The condition is worse at the menstrual periods and in cold weather. The knee-jerks are brisk; all the muscles react readily to weak faradic currents. There is no affection of sensation.

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TABLE OF THE MORE IMPORTANT SYMPTOMS IN SIXTY CASES OF MYASTHENIA GRAVIS.

		Age.	Sex.	Neurotic Heredity.	Exciting Cause.	First Symptoms.	Variation in Symptoms.	Speech.	Ocular Palsies.	Diplopia.	Ptosis.	Mastication.	Facial Weakness.	Deglutition.	Dyspnœa.	Palate.	Larynx.	Tongue.	Neck.	Extremities.	Atrophy.	Electrical Changes.	Reflexes.	Fatal.	Mode of Death.	Duration.	Post mortem.	Autopsy "Findings."
1	Wilks		F.			General weakness		X	X					X	X					X				X	Dyspnœa, sudden		X	O
2	Erb, 1st case	55	M.	O		Pain in neck and head, weakness of neck muscles				X	X			X				X	X	X	Trapez. and neck muscles	Trap., splenii and masseters dim.		O		A few months nearly well		
3	Erb, 2nd case	30	F.			Twitching in face, diplopia, ptosis, difficulty in mastication	X	X	X	X	X	X	X	X		X		X	X	X	Masseters			X	Sudden	8 months	O	
4	Erb, 3rd case	47	M.			Occipital headache and weakness in neck	X			X	X	X	X	O		X		X	X									
5	Erb, 4th case	48	M.			Occiput headache, lips stiff, diplopia		X		X	X	X		X				X	X	X	Atrophy and fibrillary tremors in tongue			O				
6	Eisenlohr	18	F.			Diplopia	X		Very little movement; X	X	X	X	X	X	X	X		X	X	X				X		2 years	X	X
7	Oppenheim	29	F.			Weakness in hands	X	X				X	X	X	X	X		X	X	X		O	Dim. excit. in deltoids and biceps		X	Sudden, dyspnœic attack with pneumonia	2½ years		
8	Shaw	37	M.		Severe diarrhœa for 14 days	Jaws, arms and legs	X	X		X	X	X	X	X	X	O		X		X				Brisk; spurious right ankle clonus	X	Dyspnœa	6 months	X	O
9	Bernhardt	44	M.			Diplopia	X	X	X	X	X	X	X	X	X		Right vocal cord paretic		X	X			Slight diminution in neck muscles		X	Sudden	4 years	O	
10	Jolly, 1st case	15	M.	O		Weakness in legs		X	O		X	X		X						X			Myasthenic reaction	Brisk	X	Sudden choking	15 months	X	O
11	Jolly, 2nd case	14	M.			Ptosis		X			X	X		O				O	X	X					O		1½ years		
12	Senator, 1st case (? case)	41	M.		Feverish for several weeks	Nasal speech	X	X				X	X	X		X		X		X			Diminished in left face	Active	X	Exhaustion	4 months	X	O
13	Senator, 2nd case (? case)	36	F.		Very amæmic, myelomata of ribs, albumosuria	Pains in head, back and legs, plus fatigue								X				Almost complete					Diminished in tongue		X	Sudden		X	O
14	Hoppe	40	M.	O		Ptosis		X	X		X	X	X	X		X	Paralysis of adductors and abductors	X		X		O			X		About 1 year	X	O
15	Remak	12	F.		Influenza and otitis media 5 months previously	Speech indistinct, eyes did not close when asleep	X	X					X	X	X	X		X		X					X	Dyspnœa, sudden	9 months	O	
16	Goldflam, 1st case	25	M.			Headache, giddiness, limitation of head movements	X	X				X		X	O	X		O	X	Especially muscles nearest trunk		O	Exhaustible; brisk	O		15 months		
17	Goldflam, 2nd case	25	M.			Weakness of extremities	X					X	X		X	O		O	X	Especially the large muscles		O	Brisk knee-jerks; slight ankle clonus	O		2 years		
18	Goldflam, 3rd case	22	F.	F.—Brain tumour M.—Congenital divergent strabismus	Anæmic	Headache, photophobia, diplopia		X	O		X	X	X	X	X	X		X		X	Especially deltoid; X		Very active	O		5 months		
19	Dreschfeld	36	F.			Double ptosis	X	X	X		X	X	X	X	X		X	Weakness of abductors	X		X	Sternomastoid trapezius and deltoid, also tongue somewhat atrophied			X	Dyspnœa	15 years	X	O
20	Suckling, 1st case	21	F.	B.—Epilepsy S.—Paralysis	Overwork	Weakness of extremities			X	All restricted nystagmus; X	X	X	X	X	X	X	O		X		Especially deltoid; X	O		Brisk	O		3 years		
21	Suckling, 2nd case	18	F.	Neurotic family history					X	All rest; X	X	X	Jaw drops X		X	O				X		O		Brisk	O		3 years		
22	Wernicke and Toby Cohn	16	F.						O	Ophthalmoplegia externa; X		X	X	X	X		Anæsthesia larynx and pharynx	X	X	X		Myasthenic reaction			X	Asphyxia, sudden		X	X
23	Mayer						X	X				X	X		X					X		O		X	Choking		X	X
24	Strümpell	21	F.			Difficulty in speaking, ptosis		X		X	X	X	X	X	X					X			Knee-jerks not exhaustible but diminish slightly	X	Sudden, dyspnœa pneumonia	19 months	X	O
25	Murri						X																Myasthenic reaction					
26	Charcot and Marinesco	13	M.			Ophthal. ext.			Complete ophthalmoplegia externa				X							Legs complete; X			Diminished excitability to both		X	Bulbar symptoms		X	X
27	Pineles	25	M.			Headache, giddiness, diplopia, right ptosis	X	X		X	X	X		X	X							O		Brisk					
28	Collins	27	F.		Six months pregnant	Weakness of extremities	X	X		X	X	X	X	X	X	X		X		X			O	Knee-jerks variable, can be tired out	O				
29	Fajersztajn	23	M.		Caught cold 10 days previously	Weakness in legs, difficulty in chewing, swallowing and speaking	X	X			X	X	X	X	X			X		X			O		O		6 months		
30	Silbermark	34	M.	O		Weakness in fingers	X	X	O		X	X	X	X	X	O		X		X					O		1½ years		
31	Devie and Roux (? case)	43	F.				X		Eyes practically immobile, slight nystagmus	X	X		Right face X										O	O				
32	Kojewnikov, 1st case	54	M.		Chill	Lips weak, difficulty in chewing and swallowing, and nasal speech	X	X	X	X	X	X	X	X	X			X			Face and masseters		O	X	Sudden, paralysis of respiration	2½ years	O	
33	Kojewnikov, 2nd case	17	F.	B.—Spasmodic asthma	Onset of menstruation	Nasal speech	X	X	X	X	X	X	X	X	X Sometimes during sleep	X	Defective adduction	X	X	X Shoulders and hips	Slight of tongue	Diminished in face. Reaction of degeneration in tongue		O		7 years		
34	Toby Cohn	19	F.	O			X	X		X	X	X	X		X			X	X	X		Myasthenic reaction		O		1 year		
35	Kalischer (? case)	64	M.			Double ptosis, paralysis of external ocular muscles, pupils sluggish														X		Some diminution to faradism	Increased	X	Paralysis of respiration		X	X
36	Widal and Marinesco	31	M.			Severe headache, ptosis, diplopia, nasal speech, &c.	X	X	X	X	X	X	X	X	X	X		X	X	X				X	Sudden	14 days	X	X
37	Berkeley	28	M.	O																X		Myasthenic reaction		X	Sudden, asphyxia		X	O
38	Schlesinger (? case)	29	F.			Difficulty in swallowing		X	O	O					O	O	O	X		X	General muscular atrophy, especially post. part of deltoid, thenar and hypothenar eminences		Arm-jerks absent; knee-jerks obtainable	X	Paralysis of diaphragm	3 months	X	O
39	Brissaud and Lantzenberg (? case)	41	M.		A cardiac affection, (?) myocarditis preceded symptoms	General wasting, paralysis of tongue and right face, diplopia	X	X	X	X	X	X	X	X	O			X	X	X	Atrophy of sternomastoid and neck muscles, also in arms and legs, and occasional fibrillation	No myasthenic reaction	Knee-jerks diminished	O				
40	Karplus	24	F.	O		Ptosis, paralysis of external eye muscle, &c.	X		X		X	X	X							X				O		2 years		
41	Eulenberg	28	M.	O	Inflammation of throat and a diphtheritic-like membrane a few months before	Diplopia and right-sided ptosis	X		Complete ophthal. ext., pupils react sluggishly			X	X	X	X	X				X		Myasthenic reaction		O		3 years		
42	Mailhouse (? case)	23	M.	O	Slight attack of diarrhœa	Weakness in neck, difficulty in swallowing, indistinct speech, ptosis	X	X	O		X	X	X	X	O	O			X	X		Myasthenic reaction		O	X	Sudden, difficulty in swallowing (? choking)	1 month	O	
43	Laquer, 1st case	21	F.		Anæmia, confined 3 months previously	Ptosis and diplopia	X	X	X	X	X		X	X	X	X	O		X	X			Increased		X	Sudden	1½ years	X	O
44	Laquer, 2nd case	49	M.	O	Frequent attacks of gout	Weakness and pain right arm and leg		X	X	X	X	X	X	X	X		O			X	Atrophy of deltoid, biceps, pectorals, interossei, calf muscles, &c.	Myasthenic reaction	Brisk; cannot be exhausted	O		6 months		
45	Wheaton	33	F.	O	Emotion (?)	Headache, photophobia and ptosis	X				X	X		X	X				X	X			Brisk	X	Dyspnœa	2 months	O	
46	Saenger	22	F.		Headache and sore throat 4 weeks previously	Difficulty in swallowing and diplopia	X	X	X	X	X		X	X	X	X			X	X		O		O				
47	Montesano	17	M.		After cold bath	Weakness of legs			X	X			X					X		X			Myasthenic reaction; diminished		O			
48	Kostetzki	24	M.																									

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Sex.	Neurotic Heredity.	Exciting Cause.	First Symptoms.	Variation in Symptoms.	Speech.	Ocular Palsies.	Diplopia.	Ptosis.	Mastication.	Facial Weakness.	Deglutition.	Dyspnœa.	Palate.	Larynx.	Tongue.	Neck.	Extremities.	Atrophy.	Electrical Changes.	Reflexes.	Fatal.	Mode of Death.	Duration.	Post mortem.	Autopsy "Findings."
F.	.. .. ..	.. ..	General weakness	..	X	X	..	..	..	..	X	X	..	..	..	..	X	.. ..	.. ..	.. ..	X	Dyspnœa, sudden ..	.. ..	X	O
M.	O	.. ..	Pain in neck and head, weakness of neck muscles	..	..	.. .. ..	..	X	X	..	X	..	..	..	X	X	X	Trapez. and neck muscles ..	Trap., splenii and masseters dim.	.. ..	O	.. ..	A few months nearly well		
F.	.. .. ..	.. ..	Twitching in face, diplopia, ptosis, difficulty in mastication	X	X	X	X	X	X	X	X	..	X	..	..	X	X	Masseters	.. ..	.. ..	X	Sudden	8 months ..	O	
M.	.. .. ..	.. ..	Occipital headache and weakness in neck	X	..	.. .. ..	..	X	X	X	O	..	X	..	..	X	X								
M.	.. .. ..	.. ..	Occiput headache, lips stiff, diplopia	..	X	.. .. ..	X	X	X	..	X	..	..	..	X	X	X	Atrophy and fibrillary tremors in tongue	.. ..	.. ..	O				
F.	.. .. ..	.. ..	Diplopia	X	..	Very little movement; X	X	X	X	X	X	X	X	..	X	X	X	.. ..	.. ..	.. ..	X	.. ..	2 years ..	X	X
F.	.. .. ..	.. ..	Weakness in hands	X	X	.. .. ..	..	..	X	X	X	X	X	..	X	X	X	O	Dim. excit. in deltoids and biceps	.. ..	X	Sudden, dyspnœic attack with pneumonia	2½ years		
M.	.. .. ..	Severe diarrhœa for 14 days ..	Jaws, arms and legs	X	X	.. .. ..	X	..	X	X	X	X	O	..	X	..	X	.. ..	.. ..	Brisk; spurious right ankle clonus	X	Dyspnœa	6 months ..	X	O
M.	.. .. ..	.. ..	Diplopia	X	X	X	X	X	X	X	X	X	..	Right vocal cord paretic	..	X	X	.. ..	Slight diminution in neck muscles	.. ..	X	Sudden	4 years ..	O	
M.	O	.. ..	Weakness in legs	..	X	O	..	X	X	..	X	..	..	..	..	..	X	.. ..	Myasthenic reaction	Brisk	X	Sudden choking ..	15 months ..	X	O
M.	.. .. ..	.. ..	Ptosis	..	X	.. .. ..	..	..	X	X	O	..	..	..	O	X	X	.. ..	.. ..	.. ..	O	.. ..	1½ years		
M.	.. .. ..	Feverish for several weeks ..	Nasal speech	X	X	.. .. ..	..	..	X	X	X	..	X	..	X	..	X	.. ..	Diminished in left face ..	Active	X	Exhaustion	4 months ..	X	O
F.	.. .. ..	Very amæmic, myelomata of ribs, albumosuria	Pains in head, back and legs, plus fatigue	..	..	.. .. ..	..	..	..	..	X	..	..	..	Almost complete X	..	.. ..	.. ..	Diminished in tongue ..	.. ..	X	Sudden	.. ..	X	O
M.	O	.. ..	Ptosis	..	X	X	..	X	X	X	X	..	X	Paralysis of adductors and abductors	..	..	X	O	O	.. ..	X	.. ..	About 1 year	X	O
F.	.. .. ..	Influenza and otitis media 5 months previously	Speech indistinct, eyes did not close when asleep	X	X	.. .. ..	..	..	..	X	X	X	X	..	X	..	X	.. ..	.. ..	.. ..	X	Dyspnœa, sudden ..	9 months ..	O	
M.	.. .. ..	.. ..	Headache, giddiness, limitation of head movements	X	X	.. .. ..	..	..	X	..	X	O	X	..	O	X	Especially muscles nearest trunk	O	O	Exhaustible; brisk ..	O	.. ..	15 months		
M.	.. .. ..	.. ..	Weakness of extremities	X	..	.. .. ..	..	..	X	X	..	X	O	..	O	X	Especially the large muscles	.. ..	O	Brisk knee-jerks; slight ankle clonus	O	.. ..	2 years		
F.	F.—Brain tumour M.—Congenital divergent strabismus	Anæmic	Headache, photophobia, diplopia ..	..	X	O	X	X	X	X	X	X	X	..	..	X	Especially deltoid; X	.. ..	.. ..	Very active	O	.. ..	5 months		
F.	.. .. ..	.. ..	Double ptosis	X	X	X	X	X	X	X	X	..	X	Weakness of abductors	X	..	X	Sternomastoid trapezius and deltoid, also tongue somewhat atrophied	.. ..	.. ..	X	Dyspnœa	15 years ..	X	O
F.	B.—Epilepsy S.—Paralysis	Overwork	Weakness of extremities	..	X	All restricted nystagmus; X	X	X	X	X	X	X	O	..	X	..	Especially deltoid; X	O	O	Brisk	O	.. ..	3 years		
F.	Neurotic family history	.. ..	.. ..	..	X	All rest; X ..	X	X	Jaw drops X	..	X	O	..	..	..	..	X	.. ..	O	Brisk	O	.. ..	3 years		
F.	.. .. ..	.. ..	.. ..	..	O	Ophthalmoplegia externa; X	..	X	X	X	X	..	X	Anæsthesia larynx and pharynx	X	X	X	.. ..	Myasthenic reaction	.. ..	X	Asphyxia, sudden ..	.. ..	X	X
..	.. .. ..	.. ..	.. ..	X	X	.. .. ..	..	X	X	..	X	..	..	..	..	..	X	O	.. ..	O	X	Choking	.. ..	X	X
F.	.. .. ..	.. ..	Difficulty in speaking, ptosis ..	..	X	.. .. ..	X	X	X	X	X	X	..	..	..	..	X	.. ..	.. ..	Knee-jerks not exhaustible but diminish slightly	X	Sudden, dyspnœa, pneumonia	19 months ..	X	O
..	.. .. ..	.. ..	.. ..	X	..	..	..	..	..	..	..	..	..	..	..	..	.. .. ..	.. ..	Myasthenic reaction						
M.	.. .. ..	.. ..	Ophthal. ext.	..	..	Complete ophthalmoplegia externa	..	..	..	X	..	..	..	..	..	..	Legs complete; X..	.. ..	Diminished excitability to both..	.. ..	X	Bulbar symptoms ..	.. ..	X	X
M.	.. .. ..	.. ..	Headache, giddiness, diplopia, right ptosis	X	X	.. .. ..	X	X	X	..	X	X	..	..	..	..	.. .. ..	O	O	Brisk					
F.	.. .. ..	Six months pregnant ..	Weakness of extremities	X	X	.. .. ..	X	X	X	X	X	X	X	..	X	..	X	.. ..	O	Knee-jerks variable, can be tired out	O				
M.	.. .. ..	Caught cold 10 days previously	Weakness in legs, difficulty in chewing, swallowing and speaking	X	X	Slight lateral nystagmus	..	X	X	X	X	..	..	..	X	X	X	.. ..	O	O	O	.. ..	6 months		
M.	O	.. ..	Weakness in fingers	X	X	O	..	X	X	X	X	..	..	..	..	O	X	.. ..	.. ..	.. ..	O	.. ..	1½ years		
F.	.. .. ..	.. ..	.. ..	X	..	Eyes practically immobile, slight nystagmus	X	X	..	Right face X	..	..	..	..	..	X	.. .. ..	.. ..	.. ..	O	O				
M.	.. .. ..	Chill	Lips weak, difficulty in chewing and swallowing, and nasal speech	X	X	X	X	X	X	X	X	X	..	..	X	..	.. .. ..	Face and masseters.. ..	O	O	X	Sudden, paralysis of respiration	2½ years ..	O	
F.	B.—Spasmodic asthma	Onset of menstruation ..	Nasal speech	X	X	X	X	X	X	X	X	Sometimes during sleep	X	Defective adduction	X	X	X Shoulders and hips	Slight of tongue	Diminished in face. Reaction of degeneration in tongue	O	O	.. ..	7 years		
F.	O	.. ..	.. ..	X	X	.. .. ..	..	X	X	X	X	..	X	..	X	X	X	.. ..	Myasthenic reaction	.. ..	O	.. ..	1 year		
M.	.. .. ..	.. ..	Double ptosis, paralysis of external ocular muscles, pupils sluggish	..	..	.. .. ..	..	..	..	..	..	..	..	..	..	..	X	O	Some diminution to faradism ..	Increased	X	Paralysis of respiration ..	.. ..	X	X
M.	.. .. ..	.. ..	Severe headache, ptosis, diplopia, nasal speech, &c.	X	X	X	X	X	X	X	X	X	X	..	..	X	X	.. ..	.. ..	.. ..	X	Sudden	14 days ..	X	X
M.	O	.. ..	.. ..	..	X	Practically immobile; X	..	X	X	X	..	..	X	..	..	X	X	O	Myasthenic reaction	X	X	Sudden, asphyxia	.. ..	X	O
F.	.. .. ..	.. ..	Difficulty in swallowing	..	X	O	O	..	..	O	..	..	..	O	O	O	X	General muscular atrophy, especially post. part of deltoid, thenar and hypothenar eminences	.. ..	Arm-jerks absent; knee-jerks obtainable	X	Paralysis of diaphragm	3 months ..	X	O
M.	.. .. ..	A cardiac affection, (?) myocarditis preceded symptoms	General wasting, paralysis of tongue and right face, diplopia	X	X	X	X	X	X	X	X	O	..	..	..	X	X	X	Atrophy of sternomastoid and neck muscles, also in arms and legs, and occasional fibrillation	No myasthenic reaction ..	Knee-jerks diminished ..	O				
F.	O	.. ..	Ptosis, paralysis of external eye muscle, &c.	X	..	X	..	X	X	X	..	..	..	..	..	..	X	.. ..	.. ..	.. ..	O	.. ..	2 years		
F.	O	Inflammation of throat and a diphtheritic-like membrane a few months before	Diplopia and right-sided ptosis ..	X	..	Complete ophthal. ext., pupils react sluggishly	..	..	X	X	X	..	X	..	..	..	X	O	Myasthenic reaction	O		.. ..	3 years		
F.	O	Slight attack of diarrhœa ..	Weakness in neck, difficulty in swallowing, indistinct speech, ptosis	X	X	O	..	X	X	X	X	O	O	..	..	X	X	O	.. ..	O	X	Sudden, difficulty in swallowing (? choking)	1 month ..	O	
..	.. .. ..	Anæmia, confined 3 months previously	Ptosis and diplopia	X	X	X	X	X	..	X	X	..	X	O	..	..	X	.. ..	.. ..	Increased	X	Sudden	1½ years ..	X	O
F.	O	Frequent attacks of gout ..	Weakness and pain right arm and leg	..	X	X	X	X	X	X	X	..	..	O	..	..	X	Atrophy of deltoid, biceps, pectorals, interossei, calf muscles, &c.	Myasthenic reaction	Brisk; cannot be exhausted	O	.. ..	6 months		
F.	O	Emotion (?)	Headache, photophobia and ptosis	X	..	.. .. ..	..	X	X	..	X	X	..	..	..	X	X	O	.. ..	Brisk	X	Dyspnœa	2 months ..	O	
..	.. ..	Headache and sore throat 4 weeks previously	Difficulty in swallowing and diplopia	X	X	X	X	X	..	X	X	..	X	..	..	X	X	.. ..	O	O	O				
..	.. ..	After cold bath	Weakness of legs	..	..	X	X	..	..	X	..	..	..	..	X	..	X	.. ..	Myasthenic reaction; diminished in left tongue and right hypo-	.. ..	O				
..	.. ..	.. ..	Weakness extremities and trunk,	X	X	.. .. ..	X	X	X	X	X	X	X	X	X	X	X								

| | | |

..	37	M.	..	..	..	Severe diarrhœa for 14 days ..	Jaws, arms and legs	X	X	..	..	X	X	X	X	X	X	X	X	0	..	X	..	X	..	..	..	..	..	..	Brisk; spurious right ankle clonus	X	With pneumonia Dyspnœa	6 months ..	X	O
..	44	M.	..	..	..	..	Diplopia	X	X	X	X	X	X	X	X	X	..	..	Right vocal cord paretic	..	X	X	..	..	..	..	Slight diminution in neck muscles	..	..	..	..	X	Sudden	4 years ..	O	
..	15	M.		O		..	Weakness in legs	..	X		O	..	X	X	..	X	..	..	..	..	..	X	..	..	..	..	Myasthenic reaction	..	Brisk	X	Sudden choking ..	15 months ..	X	O
..	14	M.	..	..	..	..	Ptosis	..	X	..	..	..	..	X	X	O	..	..	..	O	X	X	..	..	..	..	..	..	..	..	O	.. ..	1½ years				
case)	41	M.	..	..	..	Feverish for several weeks ..	Nasal speech	X	X	..	..	..	..	X	X	X	..	X	..	X	..	X	..	..	..	..	Diminished in left face ..	Active	X	Exhaustion	4 months ..	X	O				
case)	36	F.	..	..	..	Very amæmic, myelomata of ribs, albumosuria	Pains in head, back and legs, plus fatigue	..	..	..	..	..	..	..	..	X	..	..	..	Almost complete X	..	..	..	..	..	..	Diminished in tongue ..	.. ..	X	Sudden	.. ..	X	O				
..	40	M.		O		..	Ptosis	..	X		X	..	X	X	X	X	..	X	Paralysis of adductors and abductors	..	..	X		O		O	.. ..	X	About 1 year	X	O						
..	12	F.	..	..	..	Influenza and otitis media 5 months previously	Speech indistinct, eyes did not close when asleep	X	X	..	..	..	..	..	X	X	X	X	..	X	..	X	..	..	..	..	..	.. ..	X	Dyspnœa, sudden ..	9 months ..	O						
..	25	M.	..	..	..	..	Headache, giddiness, limitation of head movements	X	X	..	..	..	..	X	..	X	O	X	..	O	X	Especially muscles nearest trunk		O		O		Exhaustible; brisk ..	O	.. ..	15 months						
..	25	M.	..	..	..	..	Weakness of extremities	X	..	..	..	..	..	X	X	..	X	O	..	O	X	Especially the large muscles	..	..	..	..	O		Brisk knee-jerks; slight ankle clonus	O	.. ..	2 years						
..	22	F.	F.—Brain tumour M.—Congenital divergent strabismus			Anæmic	Headache, photophobia, diplopia ..	..	X		O	X	X	X	X	X	X	..	..	..	X	Especially deltoid; X	..	..	..	..	..		Very active	O	.. ..	5 months						
..	36	F.	..	..	..	..	Double ptosis	X	X		X	X	X	X	X	X	..	X	Weakness of abductors	X	..	X		Sternomastoid trapezius and deltoid, also tongue somewhat atrophied		..	..	..	..	X	Dyspnœa	15 years ..	X	O				
..	21	F.	B.—Epilepsy S.—Paralysis			Overwork	Weakness of extremities	..	X	All restricted nystagmus; X		X	X	X	X	X	X	O	..	X	..	Especially deltoid; X		O		Brisk	O	.. ..	3 years						
..	18	F.	Neurotic family history			..	.. ..	..	X	All rest; X ..		X	X	Jaw drops X	..	X	O	..	..	..	..	X	..	..	..	O		Brisk	O	.. ..	3 years						
ohn	16	F.	..	..	..	..	.. ..	..	O	Ophthalmoplegia externa; X		..	X	X	X	X	..	X	Anæsthesia larynx and pharynx	X	X	X	..	..	..	..	Myasthenic reaction ..	..	.. ..	X	Asphyxia, sudden ..	.. ..	X	X						
..	..	..	..	..	..	..	.. ..	X	X	..	.. .. ..	..	X	X	..	X	..	..	..	..	..	X		O		..	.. ..	O	X	Choking	.. ..	X	X				
..	21	F.	..	..	..	..	Difficulty in speaking, ptosis	..	X	..	.. ..	X	X	X	X	X	X	..	..	..	..	X	..	..	..	..	.. ..	Knee-jerks not exhaustible but diminish slightly	X	Sudden, dyspnœa pneumonia	19 months ..	X	O				
..	..	..	..	..	..	..	.. ..	X	..		..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.. .. ..	Myasthenic reaction									
o	13	M.	..	..	..	..	Ophthal. ext.	..	..	Complete ophthalmoplegia externa		..	..	..	X	..	..	..	..	..	..	..	Legs complete; X	..	..	..	..	Diminished excitability to both	.. ..	X	Bulbar symptoms ..	.. ..	X	X				
..	25	M.	..	..	..	..	Headache, giddiness, diplopia, right ptosis	X	X		.. .. ..	X	X	X	..	X	X	..	..	..	..	..	.. .. ..		O		O	Brisk									
..	27	F.	..	..	..	Six months pregnant ..	Weakness of extremities	X	X		.. .. ..	X	X	X	X	X	X	..	X	..	X	..	X	..	..	..	O	Knee-jerks variable, can be tired out	O								
..	23	M.	..	..	..	Caught cold 10 days previously	Weakness in legs, difficulty in chewing, swallowing and speaking	X	X	Slight lateral nystagmus		..	X	X	X	X	..	..	..	..	X	X	..	..	..	..	O		O	.. ..	6 months								
..	34	M.		O		..	Weakness in fingers	X	X		O	..	X	X	X	X	..	..	..	..	O	X	..	..	..	..	..	.. ..	O	.. ..	1½ years								
(?	43	F.	..	..	..	..	.. ..	X	..	Eyes practically immobile, slight nystagmus		X	X	..	Right face X	..	..	..	..	..	X	.. .. ..		..	..	..	.. ..	O		O							
..	54	M.	..	..	..	Chill	Lips weak, difficulty in chewing and swallowing, and nasal speech	X	X		X	X	X	X	X	X	X	..	..	X	..	.. .. ..		Face and masseters ..	..		O		O	X	Sudden, paralysis of respiration	2½ years ..	O						
..	17	F.	B.—Spasmodic asthma			Onset of menstruation ..	Nasal speech	X	X		X	X	X	X	X	X	Sometimes during sleep	X	Defective adduction	X	X	X	Shoulders and hips		Slight of tongue		Diminished in face. Reaction of degeneration in tongue		O		O	.. ..	7 years								
..	19	F.		O		..	.. ..	..	X	X	.. .. ..	..	X	X	X	X	..	X	..	X	X	X	..	..	..	..	Myasthenic reaction	.. ..	O	.. ..	1 year								
..	64	M.	..	..	..	..	Double ptosis, paralysis of external ocular muscles, pupils sluggish	..	..	..	.. ..	..	..	..	..	..	..	..	..	..	..	X		O		Some diminution to faradism ..		Increased	X	Paralysis of respiration	.. ..	X	X						
..	31	M.	..	..	..	..	Severe headache, ptosis, diplopia, nasal speech, &c.	X	X		X	X	X	X	X	X	..	..	..	..	..	X	..	..	..	..	..	.. ..	X	Sudden	14 days ..	X	X						
..	28	M.		O		..	.. ..	..	X	Practically immobile; X		..	X	X	X	X	..	..	..	X	X	X	..	..	..	..	Myasthenic reaction		X	X	Sudden, asphyxia ..	.. ..	X	O						
..	29	F.	..	..	..	..	Difficulty in swallowing	..	X		O	O	..	..	O	..	..	..	O	O	O	X	General muscular atrophy, especially post. part of deltoid, thenar and hypothenar eminences		.. .. ..		Arm-jerks absent; knee-jerks obtainable	X	Paralysis of diaphragm	3 months ..	X	O						
n-	41	M.	..	..	..	A cardiac affection, (?) myocarditis preceded symptoms	General wasting, paralysis of tongue and right face, diplopia	X	X		X	X	X	X	X	O	..	..	..	X	X	X	Atrophy of sternomastoid and neck muscles, also in arms and legs, and occasional fibrillation		No myasthenic reaction	..	Knee-jerks diminished ..	O									
..	24	F.		O		..	Ptosis, paralysis of external eye muscle, &c.	X	..		X	..	X	X	X	X	..	..	..	..	..	..	X	.. ..		.. ..	O	.. ..	2 years								
..	28	M.		O		Inflammation of throat and a diphtheritic-like membrane a few months before	Diplopia and right-sided ptosis ..	X	..	Complete ophthal. ext., pupils react sluggishly		..	..	X	X	X	..	X	..	..	..	X		O		Myasthenic reaction ..	..	O		.. ..	3 years								
..	2½	M.		O		Slight attack of diarrhœa ..	Weakness in neck, difficulty in swallowing, indistinct speech, ptosis	X	X		O	..	X	X	X	X	O	O	..	..	X	X		O		.. ..		O	X	Sudden, difficulty in swallowing (? choking)	1 month ..	O							
..	21	F.	..	..	..	Anæmia, confined 3 months previously	Ptosis and diplopia	X	X		X	X	X	..	X	X	..	X	O	..	..	X		.. ..		.. ..	Increased	X	Sudden	1½ years ..	X	O						
..	49	M.		O		Frequent attacks of gout ..	Weakness and pain right arm and leg	..	X		X	X	X	X	X	X	..	..	O	..	..	X		Atrophy of deltoid, biceps, pectorals, interossei, calf muscles, &c.		Myasthenic reaction ..	..	Brisk; cannot be exhausted	O	.. ..	6 months								
..	33	F.		O		Emotion (?)	Headache, photophobia and ptosis	X	..	.. .. ..		..	X	X	..	X	X	..	..	..	..	X	X		O		.. ..	Brisk	X	Dyspnœa	2 months ..	O							
..	22	F.	..	..	..	Headache and sore throat 4 weeks previously	Difficulty in swallowing and diplopia	X	X		X	X	X	..	X	X	..	X	..	..	..	X	X	.. ..		O		O	O									
..	17	M.	..	..	..	After cold bath	Weakness of legs	..	..		X	X	..	..	X	..	..	..	..	..	X	..	X	.. ..		Myasthenic reaction; diminished in left tongue and right hypothenar eminence		.. ..	O								
..	24	M.	..	..	..	..	Weakness extremities and trunk, palpitation and dyspnœa	X	X	.. .. ..		X	X	X	X	X	X	X	X	X	..	.. .. ..		In various muscles, pectorals, deltoid, thenar, hypothenar, glutei, spinæ		.. ..		O	O								
..	42	M.	..	..	..	Emotional excitement ..	Eyes, limbs, jaws, &c.	X	..	.. .. ..		..	..	..	..	X	..	..	..	..	..	.. .. ..		.. ..		No myasthenic reaction	..	O								
	28	F.	..	..	..	..	.. ..	X	X	.. .. ..		X	O	..	X	X	..	X	..	X	..	X (arms)		.. ..		O		O	O	.. ..	2 years ..							
..	37	F.	Two brothers & son have nystagmus and head tremor			Typhoid fever	Bilateral ptosis	X	X		X	X	X	X	Mask-like; no paralysis	X	..	..	..	..	X		.. ..		No myasthenic reaction	..	Not to be tired out. O		O							
..	25	F.	..	..	..	Followed birth of only child ..	Diplopia and left ptosis	..	X		X	X	X	X	X	X	..	X	Paralysis of left vocal cord	X	..	O		O		O	Slightly exaggerated		O								
..	18	F.	..	..	..	Caught cold	Tongue and jaws stiff	..	X		O	..	..	O	..	X	..	..	..	O	..	Forefinger left hand weak	.. ..		.. ..		.. ..	X	Sudden, with convulsion	Under 1 year	O							
..	..	F.	..	..	..	..	.. ..	..	X	Nystagmoid movements at times		..	X	X	..	..	..	..	..	..	..	X	.. ..		Myasthenic reaction ..	..	.. ..	O	.. ..	8 years								
..	40	M.		X		After cold baths ..	Weakness in fingers of right hand ..	X	X		X	X	X	X	X	X	X	X	X	X	X	X		O		Myasthenic reaction ..	..	Brisk; cannot be exhausted	O	.. ..	7 years								
..	24	F.		O		Anæmia, hard work ..	"Giving way" of legs	X	X		X	X	X	X	X	X	X	X	O	X	X	X		O		Myasthenic reaction ..	..	Brisk; cannot be tired out	O	.. ..	1 year								
..	44	M.		O		Acute intestinal obstruction ..	Cranial nerves and limbs	..	X	Ophthalmoplegia externa; X		..	X	X	X; right ..	X	O	O	..	X	..	X		O		O		X	O	.. ..	1 year								
..	23	F.		O		..	Difficulty in speaking	..	X		O	..	O	X	X	X	..	..	O	..	..	X		O		Myasthenic reaction		.. ..	O	.. ..	2 years								
.	25	F.	..	..	..	Chill	Ptosis	X	X		.. .. ..	X	X	X	X	..	..	..	..	X	..	X		.. ..		.. ..	.. ..	O								
)	19	F.	..	..	..	Scarlet fever	Paralysed all over	X	..	.. .. ..		..	X	..	X	..	..	O	O	O	..	O		.. ..		O		O	.. ..	3½ years							

A CASE OF ASTHENIC BULBAR PARALYSIS (MYASTHENIA PSEUDO-PARALYTICA GRAVIS).

BY ARTHUR J. HALL, M.A., M.B., M.R.C.P.

Professor of Pathology, University College, Sheffield; Hon. Physician, Sheffield Royal Hospital.

Mrs. X., a multipara, 44 years. Comes of a nervous stock, and herself suffered from migraine. Father lived to 81; mother alive, 85 years of age, healthy, but of low nervous tone. One sister has melancholia, two others have severe migraine. Has had two children, labours quite normal, last about twelve years ago. No history of syphilis. Menses regular, painful, latterly profuse. Has lately suffered a great deal from domestic worry on accouut of the presence in the house of a relative whose mental condition for the last two years has been particularly trying.

Present illness.—Early in December, 1896, the patient complained of weakness in all her muscles. She always seemed tired, and could not even hold a tea-cup for long at a time. On Christmas Day she had some swelling of the left side of the face, thought to be due to exposure to cold; at the same time slight ptosis of the left eye was noticed. The ocular muscles at this period acted perfectly regularly. After this the ptosis was frequently noticed to come on during the day and increase towards evening, whilst absent first thing in the morning. The general weakness increased, and about the beginning of February she fell down and bruised her head, apparently from weakness of her legs, as she had no vertigo. For the last few days she has remained in bed and has hardly been able to raise herself. She has some headache, no vomiting, says things appear all out of shape.

Present condition, February 11, 1897.—She lies in bed, looking languid and depressed. There is marked ptosis of the left eye and slight on the right side also. The ocular muscles act irregularly, especially in the associated movements of the left internal and right external rectus; the amount of irregularity varies from time to time, but the patient is so quickly exhausted that I found it beyond me to say exactly what muscles are

actually affected. The left internal rectus is, however, the most distinctly feeble. The grasp of the hands is feeble, but equal on the two sides. She is generally unable to stand or even to sit up by herself, but at times can both sit up and even walk quite well.

There is no wasting, no loss of sensation, no loss of control over the sphincters. The viscera seem normal in all respects.

The tendon and superficial reflexes are quite natural; there is no clonus. Speech is slow and drawling, the voice feeble as though she were tired. She has never had any choking, but often complains of being too tired to finish eating what she has begun, and complained of considerable difficulty in swallowing. The ophthalmoscopic examination was negative.

Electrical reactions perfectly normal.

I was puzzled as to the exact diagnosis. I did not find sufficient data for diagnosing any definite lesion, and was yet unwilling to call the case purely functional. At the end of a week I saw her again, and found her somewhat more cheerful and on the whole slightly improved by her rest. As she lived some distance from Sheffield we arranged that she should come into a private nursing home and try the effect of isolation, overfeeding, electricity and massage.

She came into the home towards the end of February. I saw her almost daily, and for the first two weeks she improved very greatly in every respect. She was much more cheerful, took her food well, and had less ptosis. She was also able to move herself in bed far better. In fact the result of treatment seemed most satisfactory. At this point a menstrual period came on, which lasted ten days and was very profuse in quantity. During and after this all the improvement of the past two weeks disappeared and she was just as bad as ever she had been. I hoped that this might be only temporary, but unfortunately these hopes were vain. She got steadily worse in every respect, and I felt convinced that there was some serious organic nervous disease. I asked my colleague Dr. Burgess to see her with me, which he kindly did, and we agreed that there seemed no prospect of her improving under the present treatment and that she had better go home, for she was now extremely depressed. As regards her symptoms at this date, they were as follows:—

Extreme mental depression. General weakness of all her muscles. Marked ptosis of left eye. Paresis of ocular muscles varying in extent from day to day and very difficult to determine, also the eyes frequently moved unequally when she was asked to look at any particular object. A marked nasal tone in the voice.

All these conditions were very much worse when attention was drawn to them. The patient herself noticed this, and the nurse assured me that the nasal character of the voice would be quite absent for a long period until I came, when it would begin at once. The patient also told me one morning that she had difficulty of breathing on first waking, but that it was not severe, and the nurse had not noticed any real dyspnœa. These attacks of dyspnœa were repeated two or three times.

On April 2 she went back home, and stood the journey, a distance of five miles in a carriage, very well. She slept well and seemed bright and talkative next morning. She had some difficulty in swallowing her breakfast, and after doing so she went rather blue and made a curious "rattling" in her throat. She soon recovered and seemed no worse for this. At about 1 p.m. she had some beef-tea, after which she suddenly began to have dyspnœa, became cyanosed and rapidly collapsed.

I went out immediately and at 2.30 p.m., when I saw her, the condition was as follows:—

She is lying on her back with jaw dropped; ashy gray colour, extremities blue and cold. Eyes half open, occasionally widely opened and rolling about; pupils small, equal, do not react to light. Respirations very short and spasmodic, stertorous, infrequent, cheeks drawn in at each inspiration. Pulse 100. Heart's action somewhat tumultuous. Urine passed into bed. Can articulate "Yes" and "No" when spoken to. All possible remedies were tried but she gradually became comatose and died at 5 p.m.

I was very anxious to clear up, if possible, this extremely sudden and unexpected ending, and was allowed to make an examination of the cranial contents only on April 5. This I did with the help of Drs. Burgess and Cocking, and so far as the brain and medulla oblongata were concerned a careful naked eye examination revealed absolutely nothing in any part of the membranes, vessels, or nervous tissue, which could be distinguished from that of a perfectly normal adult.

I kept the whole of the pons and medulla and portions of the cortex cerebri and cerebelli for microscopical examination. These were hardened in Müller's fluid, cut in celloidin and stained by Weigert's method, aniline blue-black, alum carmine, &c. A careful examination of a large number of sections failed to reveal any changes in the nerve-cells or fibres, or in the interstitial tissue or blood-vessels.

Reviews and Abstracts.

Ueber die Raumwahrnehmungen des Tastsinnes. By Dr. Victor Henri. Berlin. Reuther und Reichard, 1898, pp. 226.

M. Henri believes that experimental psychology has now reached a stage of development at which the critical collection of the results gained in its various departments is necessary for further progress. In the present memoir he has written a valuable account of our present knowledge of the psychology and physiology of tactile spatial perception. M. Henri is already well known for his original contributions to this subject, and his account of these make the book much more than an epitome of previous knowledge. Perhaps the most valuable feature of the book is the training which it suggests in psychological methods. The experiments described are very simple, and require very little apparatus, and that only of the simplest kind. Their careful repetition would, however, give considerable insight into the methods and characteristic features of psychological experimentation, while the experiments themselves throw light on several questions of great importance in relation to disputed psychological and philosophical problems, such as to the nature and mode of genesis of spatial perception. In the present state of psychology it is very important to separate facts from theories; nearly all psychological work is done with a bias in favour of some one school of thought, and it is often no easy matter to sift out the positive contributions to knowledge from such work. This task the author has successfully carried out. The first part of the memoir is devoted to the facts which have been more or less firmly established by experimental research, while in the second part the theories of spatial perception are reviewed in the light of these facts.

The facts are considered under two heads: (1) The perception of spatial relations by means of touch, and (2) localisation of a point on the skin. The author distinguishes sharply between these two processes, and bases this distinction partly on pathological evidence, stating that the two processes may be very differently affected in such a condition as tabes dorsalis. The

actual observations given on this head are very scanty; one case is described in which localisation on the foot was normal, while the spatial threshold was enormously increased (7-9 cm.). Further observations in this direction are needed. Of the original contributions of the author two may be especially mentioned. He has shown that the familiar illusion with crossed fingers described by Aristotle is only one example of a general law that in unusual positions of the fingers (and other parts of the body), the localisation and spatial relations of touched points are the same as if the fingers (or other parts) were in their customary positions. The second contribution is an original method of studying localisation. The ordinary method has the great disadvantage that apparent inaccuracy of localisation may be partly or wholly due to defect of the localising movement. M. Henri advocates localisation on a life-size pattern or model of the part of the body which is being investigated, and obtains satisfactory results. So far the method has been very little used in pathological cases, but with patients of ordinary intelligence should prove useful. He finds with this method that the errors of localisation tend to occur in definite directions; on the limbs a touched point is localised too far in the direction of the nearest joint. This result is ascribed to a tendency to localise in reference to certain fixed points, and the errors were found to decrease the more nearly the touched point approached such a point of reference. The observations of Mr. Victor Horsley on erroneous localisation in cortical disorders, which are very interesting in this connection, appear to have escaped the author's attention.

The author seems also to have paid insufficient attention to pressure-points. According to Goldscheider these points form the basis of tactile spatial discrimination, but the results of this worker are dismissed in a few lines as being based on minimal values, not comparable with the mean values of other observers. If Goldscheider is right, the stimulation of pressure points affects the specific nerve endings which have to do with spatial discrimination, and the existence of such specific organs is hardly a matter which can be lightly passed over in a book of this kind.

In the second part the author gives an excellent account of the various theories of spatial perception. The popular view that the perception of spatial relations is wholly dependent on movement, has, of course, as little foundation in the case of touch as in that of sight, and M. Henri points out various facts which contradict the modified view held by Wundt and others, that spatial perception depends on a psychical synthesis of tactile and muscular elements.

In Aristotle's illusion, for example, the sensations arising from the altered positions of the fingers wholly fail to influence localisation in the way they should according to this view. The author refuses to range himself with the advocates of either the genetic or nativistic views. He points out facts like the above which are irreconcilable with the genetic theories at present in the field, but, while inclining to the nativistic position he refuses to adopt it, because it has not (it probably never can have) a positive experimental basis.

W. H. R. Rivers.

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The Education of the Central Nervous System. By Reuben Post Halleck, M.A. (Yale). Pp. 258. Macmillan and Co., London and New York, 1896.

In this book attention is called to the importance of early training of the central nervous system while its brief period of plasticity lasts, and the author says his principal object in writing it is "to prescribe for our complex central nervous systems, at the proper time, the special kinds of exercise—sensory, motor, and ideational—demanded for full development." In the first chapter a short description is given of nerve-cells and nerve-fibres, the spinal cord, medulla oblongata, cerebellum, and cerebrum, including a brief account of the motor and sensory centres, and the projection, commissural, and association fibres of the brain. The fatalistic aspects of education are alluded to, and refuted by, the author, who says that he has demonstrated that young nerve-cells are more amenable to training than any other matter of which he has knowledge, and there is no doubt that careful culture can do much in children who have a healthy mental condition. If we ask how brain tracts can be modified, the answer is, through the action of sensorial impulses, motor movements, and especially by the development of the fibres of association, for without these there can be no memory, association, and motor discharge. The importance of training the faculty of attention is rightly insisted upon, and it is shown that the nerve-cells must be well nourished, in order that this faculty may be maintained at its highest point of development. As regards environment, it must afford sufficient stimuli to develop the senses, and allow for motor action in the growing nervous system. For this purpose a

life in the country is preferable to one in town. As regards age, training should begin early, but we question the author's statement that no one gets a new idea into his head after the age of thirty-three. The author rightly lays stress upon the importance of training the senses, and in chapter viii. he gives a list of objects for developing the olfactory, gustatory, auditory, and visual tracts, and the sense of touch. An interesting account of how Shakespeare's senses were trained is given, from which we learn that though he left school at the age of fourteen, he had roamed from childhood in the fields and forests that surrounded Stratford-on-Avon, and in this way developed his various senses. Passages are quoted to show that he not only observed natural objects, but human beings as well. In chapter ix. is shown the importance of recalling images of sense-objects as a means of educating the nervous system, and the fact that these images are necessary aids in further modifying and developing the sensory cells is demonstrated. The book will be useful to parents, teachers, and all who are interested in the education of children.

L'Epilepsie. Par le docteur JULES VOISIN. Pp. 420. Paris, 1897.

THIS book consists of a series of lectures given by Dr. Voisin at the Salpêtrière. The author first mentions the numerous definitions which have been given to this malady, and says, quite truly, that it is not a true disease, but a collection of symptoms which are more or less developed in one individual. He believes that heredity, which plays such an important part as a predisposing cause in the production of mental and nervous affections, scarcely acts directly in this affection, but is more active in what he calls "transformed" heredity. From a statistical table which the author gives, it appears that heredity, acting directly, only accounts for 9 per cent. of the cases, while the transformed heredity of tuberculosis and alcohol account for 28 and 31 per cent. respectively. Among the determining causes the author places sleep as the most frequent factor; and it is due to the occurrence of the fits in the night that the patient and his friends are ignorant that he suffers from them. As is well known, they occur most frequently in the early morning, owing, as the author believes, to the blood being charged with toxines. At any rate, it is a fact that when epilepsy commences, it occurs during the

night, and it is only when the patient has suffered from the affection for some time that it occurs during the day. The author proceeds to discuss the remote and immediate premonitory symptoms, the auræ, the symptoms and different varieties of the attack and their groupings, as well as the symptoms which occur after the attack, such as dulness, dementia, and epileptic mania. Dr. Voisin has noticed that in many cases there is a disturbance of the gastro-intestinal apparatus previously to the attack, and when this disappears the patient returns to his normal condition; hence he believes that a great number of cases are of infectious origin, and are due to toxines circulating in the blood. On examining it after an attack, he observed that it was not only black and pitch-like in appearance, but there was a reduction of the activity of the oxyhæmoglobin and a diminution of the red globules, besides other changes. He made a bacteriological examination of the blood, and found in two cases a staphylococcus similar to that which had previously been noticed by others, in the blood of patients who had suffered from eclampsic convulsions. He therefore thinks that the epileptic fits are produced sometimes by autointoxication and sometimes by the products of the staphylococci, which have not been sufficiently eliminated. As regards arterial tension, he noticed that it became reduced before the paroxysm and rose above the normal condition as soon as the period of stertor commenced. On examining the urine, he discovered that post-paroxysmic albuminuria existed in half the cases, and was met with in all varieties of epilepsy, that the epileptic state was always accompanied by albuminuria, but that it disappeared quickly and was very variable in quantity. He also examined the urine to ascertain its toxic character after the attack in various individuals, and noticed that the urine collected twenty-four hours after the paroxysm was of a volume considerably above the normal, urea was increased, phosphates were in excess, and peptones were almost always present. In addition, he isolated a body which was extremely toxic when injected into animals, and concluded that the urine is hypotoxic before and during the attack, and is hypertoxic afterwards. The author then goes on to discuss Jacksonian epilepsy, the spasmodic hemiplegia of infancy, the progress, diagnosis, prognosis, pathology, and treatment of epilepsy, and winds up with certain medico-legal points as to the responsibility or otherwise of patients suffering from this affection. Those who are interested in and have to treat this malady will find the book useful.

FLETCHER BEACH.

Two Cases of Hereditary Cerebellar Ataxia.—By K. Miura (Mittheilungen der Med. Facultät der Kaiserlich-Japanischen Univ. zu Tokio, Bd. iv., Heft. 1, 1898).

Case I.—Monojiro Niwa, a man, aged 37. Father died of cerebral hemorrhage; mother of hereditary cerebellar ataxia in 42nd year. Sons of a cousin of the mother said to have had the same disease. Patient's gait affected in 25th year; a temporary affection of hands in 30th year. After a febrile attack in 33rd year co-ordination worse, speech and sight involved. Increase of symptoms in 36th year caused him to stop work. On admission to hospital expression stupid, excessive facial movements in speaking, slight tremor of tongue, speech explosive, stammering, linguals specially indistinct, sputtering when speaking, pupil reaction sluggish to light and accommodation, slight horizontal nystagmus, field of vision contracted on both sides and unequally, injection of optic papillæ, with slight clouding of margins, vertebral column fairly straight, swaying of body when sitting, no muscular atrophy, imperfect co-ordination of upper and lower extremities, great difficulty in balancing on legs, reflexes unaffected, sensibility intact. The writing, a specimen of which (Japanese) is given, is said to show marked ataxia. During the few months that patient was in hospital before death supervened, symptoms of beri-beri developed, viz., lowering of sensibility in trunk and inferior limbs, sensation of pressure in epigastrium, sensitiveness to pressure in calves, vomiting, with other digestive disorders, slight œdema of legs, almost total disappearance of knee-jerks, slight rise of temperature.

The results of the autopsy are given with great detail. In the nervous system the chief points concerned the loss of bulk in certain parts, especially the pons, medulla, cerebellum, and cord. The pons was diminished in all directions, measuring 22 mm. in length, and 27 in breadth, instead of 29 and 36. The loss of bulk appeared to be due to a diminution in the transverse cerebellar fibres. The medulla oblongata was 19 mm. long by 18 mm. broad above, 16 mm. broad lower down, instead of 23, 27 and 18 respectively (according to Krause). The cerebellum weighed 80 gr. instead of 169; its volume was 72 ccm. instead of 162. (From some hitherto unpublished figures given by the author, Prof. Taguchi makes the average Japanese brain somewhat smaller than the European). No change beyond this of size could be discovered by careful naked eye and microscopic examination. The spinal cord was diminished too, being flattened

from before backwards. At the 6th cervical root it was 12 × 7 mm. instead of 12·4×9·5; at the 6th dorsal root, 8×5 instead of 8·5×7·3, and so on. No degenerative change in columns or cells, nor in the spinal roots. The complication of beri-beri accounted for degeneration of nerve fibres in peripheral nerves of the lower extremities.

Case II.—Kentei Niwa, 41 years old, elder brother of foregoing. No syphilis; alcoholic and licentious in habit. After a febrile attack in March, 1888, lasting ten days, he noticed uncertainty in gait, awkwardness of hands, affection of speech and eyesight, &c. After some improvement, he got worse in 1890-91.

Condition in June, 1896: Hearing defective, speech stammering, jerky, explosive, worse when talking to strangers, tremor of tongue when put out, tremor of chin on bringing the mouth to a point, eyeballs small, no nystagmus, reaction of pupils normal, field of vision irregularly contracted, optic discs somewhat clouded, impotence since illness began, hands tremble on being stretched out, loss of power in hands and arms, ataxia in these parts (touching points, writing), similar ataxia, only more evident, in lower limbs, "drunken" gait, reflexes not affected, diminished sensibility on outer side of leg below knee.

A year later, all the symptoms had become worse; knee-jerks nearly absent, facial muscles acting in excess in speaking or opening mouth, micturition delayed. He was still able to attend to his business of ragman.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

At the Clinical Meeting held at 11, Chandos Street, on Thursday, June 21, the following cases were shown :—

(I.) Dr. H. Head, F.R.S.—*Two cases of muscular atrophy confined to the upper extremities, probably of neuritic origin.*

(1) Mrs. C., aged 26. She was in perfect health until November, 1897. She was then six months pregnant, and a saucepan of boiling water was upset over her right hand, scalding her badly. Two months later she had a pricking feeling in the left hand which began to waste steadily. About May, 1899, one month before her last baby was born, the right hand began to waste. This wasting was preceded by a feeling of pins and needles in the hand. In August, 1899, the left hand showed marked wasting of the thenar, hypothenar and interosseous muscles. The fingers were over-extended at the metacarpo-phalangeal joints. The condition of the right hand was the same, but much slighter in degree. The reaction of the affected muscles to faradism was distinctly diminished. No other muscles in the upper extremities were affected except the lower part of the right trapezius, which appeared to be slightly wasted. When her arm was raised to the horizontal position the right scapular border projected distinctly, but the deformity disappeared immediately her arm was raised above the horizontal. In this case sensation was distinctly affected. Touch and pain were not obviously affected, but heat and cold were lost over the palm and fingers of the left hand and to a slighter degree over the palm of the right hand. The loss to cold was more certain than the loss to heat. The reflexes and pupils were normal and there were no other signs of disease. She slowly improved, but the wasting of the hands is still evident and the lower part of the trapezius appears to be slightly affected on both sides, especially on her right. Sensation is now perfect.

(2) Florence R., unmarried, aged 25. About October, 1898, she had a good deal of pain in the left supraclavicular fossa, accompanied by numbness, coldness, and "pins and needles" in the left arm and hand. About four to five months afterwards she began to notice that she dropped things from the left hand. In October, 1899, the interossei of the left hand were markedly wasted and the hand was distinctly clawed. Both thenar and hypothenar muscles were somewhat wasted and flabby but no movement of these groups was absolutely lost. There was some doubt as to whether the right hand was wasted at this time. There was distinct slight loss of sensation to heat and cold over an area on the radial border of the left forearm and hand, including the thumb. No alteration in touch or pain could be discovered. There were no other signs of disease. The pain continued in the left shoulder and back of the neck and she had occasional pain in the right arm and hand. Now both hands show marked wasting and the lower trapezius on the left side is wasted. The only loss of sensation is a slight loss to heat and cold over the tips of the thumb and forefinger of the right hand. The electrical reactions are markedly altered in the small muscles of the left hand and to a slighter extent in the lower part of the left trapezius.

(II.) Dr. COLMAN.—*Case of "ophthalmoplegic migraine."*

Ida E., aged 14. Father has had rheumatic fever. Nothing else relevant in family history. The first attack when aged 11. There was slight ptosis without pain and twitching of the eye and forehead for a day or two, then severe headache and vomiting lasting for three weeks. At the end of that time ptosis was complete. After the attack the ptosis diminished but did not pass off. There was also external squint and diplopia.

There was a second attack of headache and vomiting three months later, and several slighter ones followed, without any increase in the ptosis. Since a severe attack in December, 1898, the ptosis had been constant. The ocular movements could all be performed in the left eye, though weakly. There was slight external strabismus when at rest, the eye looking a little downward as well as out. There was diplopia on looking to the right only. The left pupil is contracted and did not react to light or dilate to shade. There was, however, wide dilatation on her attempting to look upwards. The fundus was normal. Her general health was excellent. The attacks had been slighter and much less frequent during the last eighteen months, during

which time she has taken bromide of potassium and tincture of gelsemium regularly.

(III.) Dr. D. B. Lees.—*Case* 1.—*Hæmatomyelia ?—partial recovery.*

Man, aged 47, admitted March 9, 1900. Three weeks before admission fell backwards, striking the lower part of his back. Felt severe pain and had to be assisted to walk home. Pain was in lumbar region, also between shoulders and over metacarpo-phalangeal joints of right index and middle fingers. Limbs very weak (especially right arm and leg) but not paralysed. Great effort in passing urine and fæces.

On admission, three weeks after onset.—(1) *Sensory.—Pain,* lower lumbar and sacral regions; also in right hand, over second and third metacarpo-phalangeal joints. *Hyperæsthesia* marked over lower lumbar and upper sacral regions. *Anæsthesia* over right hand and forearm; some over left hand also. *Complete sensory loss* (touch, pain, temperature and sense of position) over whole of both lower limbs; upper limit of anæsthesia 3 inches above symphysis pubis. (2) *Motor.—Right upper limb:* Decided wasting of interossei. No power of flexing the extended fingers, or of approximating thumb and little finger. Grasp very weak. Weakness of extensors of wrist. Some weakness of flexion of elbow. No wasting of arm or forearm. *Left upper:* Some wasting of interossei, but can flex extended fingers. Grasp stronger than right. Slight weakness of extension of wrist. *Right lower:* Weakness of all movements; no wasting. Right foot in position of talipes equines; cannot be dorsiflexed. *Left lower:* Stronger than right, and foot can be dorsiflexed. No wasting. *Reflexes:* Knee-jerks very exaggerated, especially right. Suprapatellar jerks very marked. Ankle clonus on both sides, especially right. *Eyes:* No paralysis of external ocular muscles. Pupils equal, react to light and with accommodation. Discs normal. *Syphilis (?):* Had gonorrhœa and bubo in right groin at 16. Scar in right groin. Two white scars with pigmented margins on left leg.

Treatment.—Pot. iod. gr. 20, liq. hyd. perchlor., ʒj. t.d.s.

March 16 (*one week after admission*).—Improved; sensation over lower limbs restored, with a feeling of "pins and needles" in them. A touch or pin-prick at any part of lower limbs or trunk (up to second rib) causes a burning sensation. Anæsthesia now limited to skin over three middle metacarpo-phalangeal joints (dorsal and palmar aspects) of right hand

and over whole of index of left hand. Inco-ordination of right upper limb still present. Knee-jerks and ankle clonus less. Plantar reflexes not obtained. Much more power in lower limbs; still cannot dorsiflex right ankle. Slight increase of power in right upper limb. Can flex extended fingers. Able now to write. Still difficulty in urination and defæcation. No bedsores. Slight ptosis and some myosis on right side noticed on one occasion. Some distension of abdomen, with dyspeptic symptoms and occasional vomiting. Some frontal headache and disturbance of vision.

April 3.—Discharged; could walk feebly with a stick, but gait spastic.

June 8.—All four limbs weaker than in April. Left grasp weak; right very weak; some tremor now on effort to squeeze. Claw-hand on right side, slightly on left. Interossei wasted. Cannot separate fingers on right side; can do so feebly on left side. Cannot touch tip of nose accurately with either index when eyes closed. Dorsiflexion of right ankle still absent, of left weak. Gait feeble and spastic. Plantar reflex generally absent, but extensor response obtained once. No ankle clonus now. Knee-jerks much less exaggerated. Sensation to pain impaired and numb over both lower limbs and up to umbilicus in front and to iliac crests and upper sacrum behind; also on radial border of right upper limb and over the first four digits; loss of temperature sense over the same areas. Touch can be felt except over the soles. Over the trunk tactile sensation is less acute, pain sensation less acute on the right side than on left. Electrical reactions normal.

Distinct ptosis and myosis on right side (variable). Visual fields apparently normal, but in the outer half of the right visual field a finger seems to him to "shake."

Case 2.—Hysterical contracture of twelve years' duration, rapidly improving under treatment.

(IV.) Dr. Leonard Guthrie.—*A mild case of Thomsen's disease.*

Boy, aged 11. Duration of symptoms twelve months. Metacarpal bones of both thumbs adducted, first and second phalanges extended and fingers flexed at metacarpo-phalangeal joints. First joints extended, terminals flexed. On grasping an object could not let go easily. The upper limbs became temporarily rigid on voluntary movement after a period of rest, but the rigidity

speedily passed off when a few active movements had taken place. Similar temporary rigidity affected the lower limbs, but was only noticeable after rest. He complained that his legs felt stiff on starting to walk. The position of the hands was constant, even during sleep. It somewhat resembled that of tetany. The boy was intelligent and not neurotic. There was no family history of Thomsen's disease.

(V.) Dr. Newton Pitt.

(1) *Case of insular sclerosis.*—Wm. T., aged 20. Liable to periodic bilious attacks. Since June, 1899, he noticed he could not read small print on account of the constant oscillation of his eyeballs. In October anæsthesia with analgesia up to the groins suddenly developed, and three times within a week he was sent home as intoxicated on account of his ataxic gait. His legs tired readily and were rather stiff. When seen in October, 1899, he had an unsteady, feeble, slightly spastic gait which soon improved considerably. The area of anæsthesia also diminished and finally disappeared. The knee-jerks were exaggerated and there was an extensor plantar reflex. There was partial optic atrophy, especially in the outer part of the discs, and more marked in the right eye, vision $\frac{6}{60}$. No ocular paralysis. Slight constant tremors of head when unsupported. Speech slightly monotonous but hardly scanning.

(2) Ed. A., aged 40. Eighteen months ago anæsthesia of second division of right fifth cranial nerve. Thirteen months ago, after an attack of gout, his gait became very unsteady, the right leg was weaker than the left. When tired he has noticed he sometimes has had diplopia with the image displaced laterally. When seen in January, 1900, no diplopia, optic discs, fields of vision, pupils and speech normal. He is so unsteady in his gait, which is also somewhat spastic, that he moves about with great difficulty. The right grasp is weaker than the left and he could not touch his nose when his eyes were shut; this has gradually improved. In bed his legs not infrequently twitch involuntarily. There is pain in the lumbar and sacral region. No bladder nor rectal trouble. Very brisk knee-jerks with extensor plantar reflex. Clonus often comes on when he attempts to walk, but with physostigma this diminished and his gait has become less spastic. During the past five months no marked changes have taken place.

(*Additional note, June* 21, 1900.)

He still has occasional diplopia, due to weakness of the right external rectus. There is now some early optic atrophy in the right disc on the outer part, and possibly some in the left eye. His vision is rather defective, he has slight hypermetropic astigmatism, but even when this is corrected his vision is about $\frac{6}{9}$. He had numbness in the right thumb for two months, so that he was unable to button his coat; this has now passed off. The diagnosis appears to lie between insular sclerosis and combined paraplegia.

(3) Nicholas R., aged 37. Five years ago he had numbness of his fingers with a squint which lasted for a month. In January, 1900, pain in sacrum, which has continued, numbness in legs, thighs, and fingers. In March his gait became unsteady and has continued to get worse. He staggers in his gait, throws up his legs erratically, especially the left, but there is some spasticity. Knee-jerks increased. Speech slovenly and tremulous. When seen in April he had numbness with anæsthesia of fingers, especially of left hand; this has now passed off. Extensor plantar reflex, but feeble in right side. He in April was unable to touch his nose with his right hand with his eyes shut; this defect has passed off. At the onset of the attack retention of urine. Paralysis of right external rectus which has greatly diminished now. Optic discs in April normal. Right pupil smaller than left; reactions normal. Slight chronic glaucoma. The defect in walking increased rapidly at first, but since April has not varied much. The anæsthesia of hands has greatly decreased.

(*Additional note, June* 21, 1900.)

The discs appear to be whiter than in the spring, and suggest an early condition of optic atrophy, but this is still indefinite. His vision is not up to the normal. The weakness of the right external rectus is much less, but there is still diplopia. The right field of vision is contracted laterally. The pupils are equal, and the tension of the eyes is normal.